



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

LAMM MEDICAL LIBRARY STAMFORD STOM.
L301 .A11 1921
Diseases of the digestive organs : with



LANE

MEDICAL



LIBRARY

COPYRIGHT
LEA & FEBIGER
1921

PRINTED IN U. S. A.

PREFACE TO THE THIRD EDITION.

THE general view expressed in the preface to the second edition of this work, that the profession at large was becoming increasingly interested in the subject of gastroenterology, has been amply confirmed by the call for a third edition. Every physician should be acquainted, if not with all the details set forth herein, at least with the fact that diseases of the digestive organs can be accurately diagnosed and the prospect of recovery thereby very materially enhanced. Moreover, while personal instruction is the ideal training method, the hope entertained by the author that the text and the illustrations accompanying it would place at the command of the practitioner a fair proportion of the results attained by special research has been more than justified by the history of the two previous editions. In the interests of medical science this demand of the profession for definite information is satisfying in a larger sense than merely an author's gratification.

Several new colored plates, roentgenograms, engravings and a considerable amount of new material have been incorporated in this edition, reflecting the progress that has been made in recent years in this branch of medicine; and several matters which could only be outlined in previous editions have been made the subject of detailed discussion.

The plan of the work, as before, follows the physiologic path of the digestive tract, beginning with diseases of the mouth, and taking up in succession the pharynx, esophagus, stomach, liver, gall bladder, bile ducts, pancreas, small intestine, vermiform appendix, cecum, colon, sigmoid flexure, rectum, and anus. The author has attempted to put before the practitioner, in an orderly, consecutive manner, the diagnosis and treatment of digestive diseases, and to make available all the resources at his command. There is an unfortunate tendency nowadays to isolate the consideration of diseases of the digestive organs from the great body of internal medicine. But there is a direct connection between the

functions of the digestive tract and the functions of other organs, and this fact of interdependence is of prime significance in gastroenterologic pathology. The author has endeavored to reaffirm the intimate relationship between gastroenterology and the other branches of internal medicine.

No subject has profited more by the modern spirit of scientific research than the diagnosis and treatment of diseases of the digestive organs. Many new methods of investigation, physical, chemical, microscopical and clinical, have sharpened our vision and given us certainty in dealing with obscure pathologic processes. Without the least inclination to discourage research, but rather with a profound desire to stimulate it, the author has striven to eliminate abstract theories and to present to the practitioner only the practical, the trustworthy and the helpful. The book is intended not only as an aid to the specialist in diseases of the digestive organs, but also as a ready reference work for the general practitioner and surgeon.

The physiology of digestion has been considered from the viewpoint of the clinician rather than from that of the physiologist; and attention has been given to recent progress in the study of internal secretions, which has contributed much to our knowledge of the physiology of digestion.

The stomach tube and the qualitative and quantitative analyses which disclose the actual condition of the gastric functions have become a necessity in the diagnosis of digestive disorders. Space has been devoted to many tests and reactions for the diagnosis of carcinoma, such as the Wolff-Junghans, Abderhalden, Gluzinski, Salomon, tryptophan, hemolytic, antitryptic, miostagmin, cyto-diagnostic, blood-sugar tolerance, and others.

By the use of the duodenal tube the contents of the duodenum have become available and subject to analysis, as in the case of the gastric contents. A chapter is devoted to the employment of the tube for draining the gall bladder and bile ducts, duodenal lavage, duodenal feeding, removal of the duodenal contents, and examination of the latter. The several duodenal tubes in use, each with its own advantages, are fully described. With reference to examination of the duodenal enzymes, their reactions in health and disease are shown in colored plates, so that the practitioner can easily check up his tests.

Recent methods of examining the feces to determine the condi-

tion of the intestinal function are indispensable to the physician who would deal successfully with affections of the digestive organs. These new procedures are particularly valuable in cases in which the subjective sensations or symptoms are indistinct or absent. With the diagnostic technic now available for the examination of the feces after a test diet, we can determine the origin, formerly obscure, of many disturbances of the intestine. The diagnostician is now able to detect the more minute disturbances of absorption, secretion and motility, and institute rational treatment. The test-diet stool findings in each one of the diseases of the digestive organs are fully explained in due order.

Roentgenography of the esophagus, stomach and intestine shows location, relations, form, dimensions and muscular functions of these organs. The Roentgen ray has brought an element of exactness into diagnosis, making exploratory laparotomy often unnecessary. One chapter, equipped with copious illustrations, is devoted to this important subject. For the roentgenograms I am indebted to Dr. P. M. Hickey and Dr. W. A. Evans, of this city.

Dietetic treatment in its various subdivisions is dealt with, and in the discussion of the treatment of each pathologic condition the respective dietary directions are given. Besides details as to the importance of the vitamins, comprehensive tables of caloric values are included in this section of the work. One chapter has been devoted to diet in typhoid fever, with special reference to the high caloric feeding now favored by many clinicians.

The principles and the details of massage of the stomach and intestine deserve special consideration. The technic of massage is minutely described and the manipulations illustrated.

Convinced of the importance of hydrotherapeutic measures the efficacy of which depends on temperature, method, timeliness, duration, etc., the author has presented them as fully as the scope of the volume permitted, and has added practicable suggestions for the use of packs, baths, compresses, and douches.

Mineral waters represent a considerable part of gastro-intestinal therapy. Though the American waters are fairly well known, they are not fully appreciated. It is not necessary that a patient be sent across the seas for something that can be secured at home. The author has given in brief compass a list of native and foreign waters available for use in gastro-intestinal affections, together with recommendations in regard to their use.

The moist heat prevailing in the mouth furnishes a most favorable condition for the growth of microorganisms. Such diseases as syphilis, tuberculosis, leukoplakia, stomatitis, and affections of the tongue, tonsils, salivary glands, salivary ducts, alveolar processes, lips and gums, make the mouth a focus of infection. During mastication the bacteria are squeezed out of the pathologic tissue and carried with the food into the stomach and intestine. Recent experiments on the lower animals prove that the intravenous injection of specific microorganisms cultivated from the tonsils and apical abscesses of the teeth may induce gastric and duodenal ulcer, cholangitis, cholecystitis, pancreatitis, appendicitis, myocarditis, arthritis, goiter, and enlarged glands. Focal infection is certainly a predominating factor in the etiology of many gastrointestinal diseases. Hence the great importance of the early detection and treatment of variations from the normal in the vestibule of the digestive tract.

The diagnosis and treatment of the many pathologic changes of the esophagus have been greatly advanced by new methods. They can now be diagnosed by means of bougies, the esophagoscope, and the Roentgen ray. Through the esophagoscope we can apply local treatment directly to the esophagus. Varieties of esophagitis, ulcers, neoplasms, strictures, spasms, diverticula, dilatation, rupture, perforation, malacia, hemorrhage, neuroses, parasites and foreign bodies, are discussed in the chapter on diseases of the esophagus.

It is often difficult to establish the diagnosis of a nervous or functional derangement of the digestive organs; an understanding of the vegetative nervous system is here of great assistance. These nerves can be stimulated and inhibited by certain medications. In connection with this subject the opposing conditions, vagotonia and sympathicotonia, are elaborated and their important signs and symptoms tabulated. Pharmacodynamic and physiologic tests for the diagnosis of disturbances of the vegetative nervous system are given in detail, so that the clinician may be enabled to employ them readily. Neuroses of the digestive organs resulting in motor disturbances, sensory disturbances, and secretory disturbances, are discussed.

Besides the several chapters on gastric disorders classed as neuroses, attention is given to gastritis, motor insufficiency, gastric ulcer, gastric and intestinal hemorrhage, erosions, perigastritis,

arteriosclerosis, syphilis, tuberculosis, and tumors of the stomach, in separate chapters.

Recent discoveries have shown that the liver and pancreas possess manifold functions in the economy of metabolism. An injury to their structure brings about a functional disturbance. The clinical ensemble of diseases of the liver, gall bladder and pancreas has been brought into clearer light, and the medical treatment broadened by rational methods. Chapters XXXIII, XXXIV and XXXV contain a summary of present-day knowledge on this subject.

In the chapter devoted to gastroenteroptosis the complications of kinks, bends, loops, redundancy, adhesions, bands, membranes, and CECUM MOBILE are considered. In addition to the physical, electrotherapeutic, hydrotherapeutic, mechanical and medicinal treatment of gastroenteroptosis, hyperalimentation is discussed in detail.

Separate chapters cover the important subjects of acute enterocolitis, chronic enterocolitis, enteritis membranacea, chronic diarrhea, and chronic constipation. The subject of chronic constipation has been divided into atonic, spastic, and fragmentary. Because of its importance, a chapter is devoted to duodenal ulcer. Catarrhal, follicular, tubercular and syphilitic ulcers of the intestine are also considered at length. Another chapter deals with chronic intestinal toxemia, chronic intestinal stasis, and ileal regurgitation, and their medical and surgical treatment. Since the accumulation of gas is a symptom in many diseases of gastrointestinal origin, a chapter on flatulence, meteorism and tympanites has become necessary; both exogenous and endogenous gases are considered and their respective treatments given.

Obstructions of the intestine due to kinks, adhesions, membranes and tumors, as well as those due to strangulation, intussusception, volvulus and foreign bodies, are considered. A chapter has been assigned to stricture of the intestine. Tumors of the intestine, appendicitis, nervous diseases of the intestine, perisigmoiditis, diverticulitis, sigmoiditis, and idiopathic dilatation of the colon are considered in separate chapters.

Uncinariasis, which is now the subject of widespread medical interest, is included in the chapter dealing with animal parasites of the intestine.

The diagnosis and treatment of diseases of the rectum and anus

The moist heat prevailing in the mouth furnishes a most favorable condition for the growth of microorganisms. Such diseases as syphilis, tuberculosis, leukoplakia, stomatitis, and affections of the tongue, tonsils, salivary glands, salivary ducts, alveolar processes, lips and gums, make the mouth a focus of infection. During mastication the bacteria are squeezed out of the pathologic tissue and carried with the food into the stomach and intestine. Recent experiments on the lower animals prove that the intravenous injection of specific microorganisms cultivated from the tonsils and apical abscesses of the teeth may induce gastric and duodenal ulcer, cholangitis, cholecystitis, pancreatitis, appendicitis, myocarditis, arthritis, goiter, and enlarged glands. Focal infection is certainly a predominating factor in the etiology of many gastrointestinal diseases. Hence the great importance of the early detection and treatment of variations from the normal in the vestibule of the digestive tract.

The diagnosis and treatment of the many pathologic changes of the esophagus have been greatly advanced by new methods. They can now be diagnosed by means of bougies, the esophagoscope, and the Roentgen ray. Through the esophagoscope we can apply local treatment directly to the esophagus. Varieties of esophagitis, ulcers, neoplasms, strictures, spasms, diverticula, dilatation, rupture, perforation, malacia, hemorrhage, neuroses, parasites and foreign bodies, are discussed in the chapter on diseases of the esophagus.

It is often difficult to establish the diagnosis of a nervous or functional derangement of the digestive organs; an understanding of the vegetative nervous system is here of great assistance. These nerves can be stimulated and inhibited by certain medications. In connection with this subject the opposing conditions, vagotonia and sympathicotonia, are elaborated and their important signs and symptoms tabulated. Pharmacodynamic and physiologic tests for the diagnosis of disturbances of the vegetative nervous system are given in detail, so that the clinician may be enabled to employ them readily. Neuroses of the digestive organs resulting in motor disturbances, sensory disturbances, and secretory disturbances, are discussed.

Besides the several chapters on gastric disorders classed as neuroses, attention is given to gastritis, motor insufficiency, gastric ulcer, gastric and intestinal hemorrhage, erosions, perigastritis,

arteriosclerosis, syphilis, tuberculosis, and tumors of the stomach, in separate chapters.

Recent discoveries have shown that the liver and pancreas possess manifold functions in the economy of metabolism. An injury to their structure brings about a functional disturbance. The clinical ensemble of diseases of the liver, gall bladder and pancreas has been brought into clearer light, and the medical treatment broadened by rational methods. Chapters XXXIII, XXXIV and XXXV contain a summary of present-day knowledge on this subject.

In the chapter devoted to gastroenteroptosis the complications of kinks, bends, loops, redundancy, adhesions, bands, membranes, and CECUM MOBILE are considered. In addition to the physical, electrotherapeutic, hydrotherapeutic, mechanical and medicinal treatment of gastroenteroptosis, hyperalimentation is discussed in detail.

Separate chapters cover the important subjects of acute enterocolitis, chronic enterocolitis, enteritis membranacea, chronic diarrhea, and chronic constipation. The subject of chronic constipation has been divided into atonic, spastic, and fragmentary. Because of its importance, a chapter is devoted to duodenal ulcer. Catarrhal, follicular, tubercular and syphilitic ulcers of the intestine are also considered at length. Another chapter deals with chronic intestinal toxemia, chronic intestinal stasis, and ileal regurgitation, and their medical and surgical treatment. Since the accumulation of gas is a symptom in many diseases of gastrointestinal origin, a chapter on flatulence, meteorism and tympanites has become necessary; both exogenous and endogenous gases are considered and their respective treatments given.

Obstructions of the intestine due to kinks, adhesions, membranes and tumors, as well as those due to strangulation, intussusception, volvulus and foreign bodies, are considered. A chapter has been assigned to stricture of the intestine. Tumors of the intestine, appendicitis, nervous diseases of the intestine, periaigmoiditis, diverticulitis, sigmoiditis, and idiopathic dilatation of the colon are considered in separate chapters.

Uncinariasis, which is now the subject of widespread medical interest, is included in the chapter dealing with animal parasites of the intestine.

The diagnosis and treatment of diseases of the rectum and anus

Intestinal Digestion—**Absorption in the Small Intestine—**

Fat Absorption	60
Carbohydrate Absorption	61
Cellulose and Hemicellulose Absorption	61
Absorption of Aqueous and Saline Solutions	61
Digestion and Absorption in the Large Intestine	61
Intestinal Movements	62
Segmenting	62
Peristaltic	62
Pendulum	63
Anastalsis	63
Keith Nodal Tissue	63
Law of Contrary Innervation	65
Feces	65

CHAPTER II.**EXAMINATION OF THE STOMACH CONTENTS.**

Stomach Contents	66
Test Meals	67
Ewald-Boss Test Breakfast	67
Boss Test Breakfast	67
Riegel Test Dinner	68
Microscopic Examination of Stomach Contents	68
Methods of Obtaining Stomach Contents	68
Expression	68
Aspiration	68
Author's Improved Stomach Tube	68
Einhorn Stomach Bucket	70
Regurgitation	71
Inspection of Stomach Contents	72
Determination of Gastric Juice	73
Color	73
Color	74
Consistency	74
Chemical Examination of Stomach Contents	74
Apparatus	74
Determination of Reaction	75
Dimethylamidoazobenzol Test	75
Ginsburg's Test	76
Quantitative Analysis of Stomach Contents	76
Normal Solutions	77
Fractional Analysis	78
Refluxes Gastroduodenal Tube	78
Test Breakfast Secretory Curve	79
Phenolphthalein Test for Total Acidity	79
Topfer's Method of Quantitative Analysis	80
Combined Hydrochloric Acid	81
Lactic Acid	82
1. Behnemann's Test	82
Strauss' Test	83

CONTENTS

11

Examination of Enzymes	83
Pepsinogen and Pepsin	83
Determination of Pepsin	83
Jacoby-Solms Test	83
Mett's Test	84
The Gelatin Test	84
Qualitative Test for Rennin	84
Test for Propeptone	85
Test for Peptone	85
Carbohydrate Digestion in the Stomach	85
Blood in the Stomach Contents	86
Weber's Guaiac Test	86
Tests for Carcinoma	86
Salomon's Test	86
Wolff and Junghans Test	87
Cytodagnosis	87
Glycyltryptophan Test	87
Gluzinski's Test	88
Indirect Methods of Gastric Analysis	88
Benedict's Effervescence Test for Acidity	88
Thread Test for Acidity	88
Friedrich's Test	89
Quantitative Acidity Test	89
Günzburg's Test for the Absorptive Power of the Stomach	89
Sahl's Desmoid Test	90
Motor Function of the Stomach	90
Leube Test Meal	90
Chlorophyl Test	91
Permeability of the Pylorus	91
Duodenal Bucket	91
Microscopic Examination of Stomach Contents	92
Changes in Gastric Secretion Due to Pathological Conditions	94
Gastric Neuroses	94
Hyperacidity; Hyperchlorhydria	94
Hypersecretion, Gastrosuccorrhea; Gastrorrhea; Gastrochylorrhea	94
Acute Gastritis	95
Chronic Gastritis	95
Achyia Gastrica	95
Motor Insufficiency (Atony and Dilatation)	95
Pyloric Stenosis	96
Pyloric Insufficiency	96
Gastric Ulcer	97
Fistulas of the Stomach	97
Gastric Carcinoma	97

CHAPTER III.

EXAMINATION OF THE DUODENAL CONTENTS.

Duodenal Tubes	98
Einhorn Duodenal Tube	98

Duodenal Tubes—	
Gross Duodenal Tube	100
Palefski Duodenal Tube	100
Jutte Duodenal Tube	100
Determination of Tube in Duodenum	100
Characteristics of Duodenal Contents	101
Urobilin and Urobilinogen	101
Test Meal	101
Examination for Enzymes	102
Starch Agar Tubes	102
Olive Oil Agar Tubes	102
Hemoglobin Agar Tubes	102
Mode of Procedure	102
Determination of Enzymes	103
Changes in Pathological Conditions	103
Eupancrustism	103
Hyperpancreatism	103
Hypopancreatism	103
Dyspancreatism	103
Heteropancreatism	103
Direct Medication	104
Draining the Gall Bladder and Bile Ducts	104
Oxygen Insufflation	105
Duodenal Lavage	105
Apparatus	106
Technic	106
Character of Irrigating Fluid	106
Indications for Duodenal Lavage	107
Rheumatism, Sciatica, etc.	107
Intestinal Stasis	107
Bacteriology of the Duodenum	107
Results of Direct Examination of Duodenal Contents	109
Gall Bladder	109
Cholecystitis	109
Bile	109
Obstruction of Common Duct	109
Bile and Pancreatic Juice	109
Pancreatitis	109
Duodenitis	110
Duodenal Ulcer	110
Typhoid Fever	110
Pernicious Anemia	110

CHAPTER IV.

EXAMINATION OF THE FECES.

Importance of This Procedure	111
The Test Diet and Its Administration	112
Examination of the Test-diet Stool	114
Apparatus	114
Macroscopic Examination	115

Examination of the Test-diet Stool—	
Microscopic Examination	115
Chemical Examination	116
Reaction Test	116
Sublimate Test	116
Incubator Test	116
Examination for Dissolved Protein	117
Bacterial Preparations	118
The Normal Test-diet Stool	118
Pathologic Stools and Their Significance in the Diagnosis of Gastric and	
Intestinal Affections	119
Pathologic Food Remnants	119
Connective Tissue	119
Muscle Remnants	120
Fat	120
Potato Remnants	121
Starch	121
Pathologic Products of the Intestinal Wall	121
Mucus	121
Soluble Protein	121
Pus	122
Unchanged Biliary Pigment (bilirubin)	122
Bacteria	122
The Demonstration of Blood in the Feces	123
Occult Blood	123
Benzidin Test for Occult Blood	123
Phenolphthalein Ring Test for Occult Blood	124
The Demonstration of Ferments in the Feces	125
Trypsin	125
The Plate Test of Müller-Schlecht	125
The Casein Method of Gross	125
Steapsin	125
Grützner-Gamgee Test	126
von Oefele Test	126
The Nuclei Test of Adolf Schmidt	126
Diastase Test (Wohlgemuth)	127
Fat Digestion Tests	127
Cannan or Charcoal Test	128
Sahl's Glutoid Capsule Test	129
Luhorn's Bead Test	129
The Test-diet Stool Findings in Gastric and Intestinal Affections	131
Achyia Gastrica and Subacidity	131
Hyperacidity	131
Gastric Ulcer and Gastric Carcinoma	131
Chronic Catarrh of the Small Intestine	131
Chronic Catarrh of the Large Intestine	132
Dysentery	133
Intestinal Tuberculosis	133
Duodenal Ulcer	133
Enteritis Membranacea	133
Atonic Constipation	133
Spastic Constipation	134

The Test-diet Stool Findings in Gastric and Intestinal Affections—

Intestinal Fermentation Dyspepsia	134
Nervous Diarrhea	134
Stenosis and Intestinal Carcinoma	134

CHAPTER V.

ROENTGEN-RAY EXAMINATION.

Technic for Roentgenographic Examination	135
Roentgen Fluoroscopy	136
Examination of the Esophagus	136
Spasm of the Esophagus	137
Cardiospasm	137
Diverticulum of the Esophagus	138
Deep-seated Diverticula	138
Carcinoma of the Esophagus	138
Examination of the Stomach	138
Gastropptosis	139
Tonus	140
Motility	140
Atony	140
Dilatation	140
Gastric Ulcer	141
Callous Ulcer	141
Perforating Ulcer (Haudek's niche)	141
Hour-glass Stomach	142
Carcinoma	142
Pyloric Obstruction	142
Examination of the Intestine	143
Duodenal Ulcer	143
Normal Duodenal Cap	143
Ileal Stasis	144
Colon	144
Normal Motility	145
Position	145
Deviation	146
Constipation and Colonic Stasis	146
Cecum Mobile	146
Vermiform Appendix	146
Colonic Stasis	147
Sigmoid Flexure	148
Diverticulitis	148
Hirschsprung's Disease	148
Stenosis	148
Multiple Diverticula	149
Carcinoma of the Rectum	149
Diagnosis of Postoperative Obstructions	149
Pancreas	149
Liver	149
Gall Bladder	149
Gallstones	150

CONTENTS

15

Examination of the Intestine—

Spleen	150
Peritoneal Inflation	150

CHAPTER VI.

DIET IN GASTRIC DISEASES.

Heat Value of Foods	151
Dietary Regulations and Lists	152
Composition of Common American Foods	153
Protein, Fat, Carbohydrates, Calories	153
Vitamin	157
Solubilities of Vitamins	158
Stability of Vitamins	159
Meat	159
Gelatin	161
Beef Tea	161
Eggs	161
Fat	162
Milk	162
Karell Cure	162
Buttermilk	164
Whey	164
Koumiss	164
Kefir	164
Yoghurt	164
Bulgarian Lactic Acid Bacilli	165
Cheese	165
Brad	165
Gruel Soups	166
Potatoes	166
Rice	166
Green Vegetables	167
Legumes	167
Fruit	167
Sugar	167
Spices	167
Water	168
Alcohol	168
Tea and Coffee	169
Coron	169
Tobacco	169
Instructing the Patient	169

CHAPTER VII.

DIET IN INTESTINAL DISEASES.

Regulation of the Diet	172
Constipating Diet	172

Diagnosis by Examination of Feces	173
Fermentation	173
Putrefaction	173
Antiseptic Food	174
Antiputrefactive Diet	174
Milk	175
Salicylic Milk	176
Gelatin	177
Whortleberries and Blackberries	177
Antibacterial Soups	178
Jellies	178
Green Vegetables	179
Beverages	180
Antifermentative Diet	181
Protein-fat	181
Watery Soups	182
Chicken, Squab, Eggs, Noodles, Zwieback, Toast	182
Laxative Diet	182
Cellulose Digestion	183
Indigestible Residues	184
Graham Bread, Rye Bread, etc.	184
Agar	184
Raw and Cooked Fruit	185

CHAPTER VIII.

ARTIFICIAL FOOD PREPARATIONS.

Preparations of Animal Protein	187
Somatose	187
Carringen	188
Tropon	188
Salvatose	188
Fersan	188
Peptones	188
Preparations from Vegetable Protein	191
Roborat	191
Aleuronat Flour	191
Mutase	191
Preparations from Milk Protein	191
Nutrose	191
Eucasin	192
Sanatogen	192
Plasmon	192
Milk Somatose	192
Globon	192
Galactogen	192
Mammula	192
Nutritive Substances from Egg Protein	192
Nutritive-Heyden	192
Protogen	192

CONTENTS

17

Preparations from Carbohydrates	192
Finely Divided Flours	193
Dextrinated Flours	193
Malt Extract	193
Malt Soup	193
Maltose Buttermilk	193
Maltine	193
Mixed Nutritive Preparations	193
Hygama	193
Odda	193
Protein-milk-salt-cocoa	193
Rachout	194
Acorn-cocoa	194
Preparations Containing Fat	194
Russell's Emulsion	194
Nutrole	194
Sevetol	194
Cod-liver Oil	194
Oil of Sesame	194
Lipamin	194
Kraft Chocolate	194
Milk Preparations	194
Vegetable Milk	194
Cream Protein Mixture	194
Fat Milks	194
Kefir and Koumiss	194
Stimulating Preparations	194
Meat Extract	194
Beef Tea	194
Meat Juice	194
Essence of Beef	195
Fluid Meat	195
Bovril	195
Karno	195
Composition and Relative Values of Meat Extracts	195

CHAPTER IX.

LAVAGE OF THE STOMACH.

Indications	197
Contra-indications	198
Apparatus	199
Technic	199
Stomach Tube and Funnel	200
Autolavage	201
Suction Bulb (Friedlieb)	203
Suction Tube (Strauss)	204
The Stomach Douche	205
Perforated Tube (Rosenheim)	205
Richter's Method	205
Einhorn's Apparatus	206
Turck's Double-flow Tube	206
Stomach Tube (Chase)	207

CHAPTER X.

MASSAGE—ELECTRICITY.

Massage of the Stomach—	
Indications	208
Contra-indications	208
Technic	208
Tapotement	209
Pétrissage	210
Crédé's Method	210
Vibratory Massage	210
Intestinal Massage	211
Technic	211
Rotating Effleurage	212
Deep Kneading	212
Rotating Pétrissage	213
Tapotement	213
Chronic Constipation	214
Vibrating Massage	214
Electric Treatment	214
Indications	215
Intraventricular Electrization	216
Stomach Electrode (Boas)	216
Stomach Electrode (Wegele)	217
Intragastric Electrode (Einhorn)	217
Combined Stomach Tube and Electrode (Stockton)	218
Extraventricular Electrization	219
The Sinusoidal Current	219

CHAPTER XI.

TREATMENT OF DISEASES OF THE INTESTINE THROUGH THE RECTUM.

Cleansing Enemata	220
Technic	220
Rectal Tube	221
Glycerin Enemata	222
Glycerin Suppositories	223
Oil Enemata	223
Oil Enemator (Zweig)	224
Oil Enemator (Roberts)	225
Paraffin Enemata	225
Carbon Dioxid	226
Hile Enemata	226
Cathartics	227
Chloroform-water Irrigations	227
Air Insufflation	228
Intestinal Douche	228
Mechanical Treatment	228
Swedish Manipulation	228
Rectal Massage	229

Mechanical Treatment—	
Rectal Tampons	230
Proctoscope (Kelly)	230
Rectal Electrodes	230
Treatment of Intestinal Irritation	232
Irrigation of the Intestine	232
Technic	232
Irrigation Tube (Zweig)	233
Irrigation Apparatus (Rosenberg)	234
Irrigation Tube (Wolbarst)	235
Antiseptic Irrigations	235
Sedative Irrigations	236
Astringent Irrigations	236
Krameria in Rectal Catarrh	236
Natural Mineral Waters	237
Dry Treatment	237
Pneumatic Sigmoidoscope (Strauss)	237
Introduction of Sigmoidoscope	238
Powder Blower (Rosenberg)	238
Proctoclysis	239
Apparatus	239
Heating Chamber for Electric Unit (Elbrecht)	240
Self-retaining Rectal Tips	240
Electric Heating Unit	241
Electric Heater in Operation	242
Heat Unit for Alcohol or Bunsen Flame	242
Alcohol or Gas Heater (Elbrecht)	243
Glass Attachment for Proctoclysis	244
Thermos Proctoclysis Apparatus	245
Nutrient Enemata	245
Technic	245
Variety	245

CHAPTER XII.

HYDROTHERAPEUTICS—MINERAL WATERS.

Hydratic and Thermic Treatment	247
Wet Rub	247
Half Baths	248
Cold Entire Pack	248
Warm Entire Pack	249
Prolonged Baths	249
Oxygen and Carbon Dioxid Baths	249
Indications for Hydrotherapeutic Treatment	249
Compresses	250
Mashed-potato Poultice	250
Linseed Meal Poultice	250
Pressnitz Bandage	250
Lester Cooling Apparatus	251
Douches	251
Mineral Waters	252

Intestinal Sedatives—

Antiseptics and Antifermentatives—

Thiocol	281
Enterol	281
Nosophen	281
Menthol	281
Resorcinol	281
Saccharin	281
Yeast	281

Purgatives	282
Indications for the Administration of Purgatives	282
Opium in Purgation	283
Benzyl Benzoate in Intestinal Spasm	284
Agar Combinations	287

CHAPTER XV.

DISEASES OF THE MOUTH.

Anatomy of the Mouth	289
Oral Sepsis	290
Focal Infection	290
General Treatment of the Diseases of the Mouth	293
Oral Affections in General Intoxications	299
From Metals	299
Mercury	299
Bismuth	300
Lead	301
Silver	301
From Metalloids	301
Phosphorus	301
Bromids	301
Iodids	301
Medicinal Exanthema	301
Affections of the Mouth in Constitutional Infectious Diseases (measles, scarlet fever, rubeola, varicella, variola, vaccinia, typhoid and paratyphoid fevers, foot-and-mouth disease, influenza)	302
Diseases of the Mouth in Non-infectious Constitutional Diseases (hemophilia, chlorosis, purpura, scorbutus, infantile scurvy, diabetes, gout)	302
Erosions and Burns of the Mouth	303
Erosions	303
Treatment	303
Burns	304
Lesions of the Oral Mucosa	304
Traumatic Lesions	304
Treatment	304
Thickening of Epithelium	304
Palatal Ulcers	304
Treatment	304
Lingual Ulcers	305
Treatment	305
Traumatic Tumors	305
Treatment	305

Stomatitis	305
Simple or Catarrhal Stomatitis	305
Treatment	305
Gangrenous Stomatitis	305
Treatment	306
Noma	306
Treatment	307
Erysipelatous Stomatitis	307
Treatment	307
Aphthæ	307
Treatment	308
Chronic Aphthæ (Aphthæ Tropicae)	308
Sprue	308
Thrush	309
Treatment	309
Syphilis of the Mouth	309
Primary Syphilis	309
Treatment	310
Secondary Syphilis	310
Treatment	311
Tertiary Syphilis	311
Treatment	312
Tuberculosis of the Mouth	312
Lupus of the Oral Mucosa	312
Treatment	313
Glanders of the Mouth	313
Treatment	314
Leprosy of the Mouth	314
Treatment	315
Scieroma	315
Treatment	315
Actinomycosis	316
Treatment	316
Skin Diseases in the Mouth	316
Eczema	317
Treatment	317
Lichen Planus	317
Treatment	317
Lupus Erythematosus	317
Treatment	317
Pemphigus	317
Treatment	318
Erythema Exudativum Multiforme	318
Herpes	318
Urticaria	318
Scleroderma	318
Leukoplakia	319
Symptoms	319
Treatment	319
Animal Parasites in the Mouth	320
Nervous Affections of the Mouth	320
Paralysis of the Facial Nerve	320

Nervous Affections of the Mouth—**Paralysis of the Facial Nerve—**

Treatment 321

Glossodynia 321

Treatment 321

Vasomotor, Trophic and Secretory Disorders 321

Affections of the Tongue 322

Malformation 322

Coating or Furring 322

Treatment 322

Lingua Geographica 323

Treatment 323

Hair-tongue (Lingua Nigra) 323

Treatment 323

Phlegmonous Processes of the Tongue 324

Abscess 324

Treatment 324

Acute Diffused Glossitis 324

Treatment 324

Decubital Ulcer of the Tongue 325

Treatment 325

Chronic Superficial Glossitis 325

Treatment 325

Acute Papular Glossitis 325

Treatment 326

Macroglossia 326

Lingua Placata 326

Affections of the Lingual Tonsil 326

Acute Lingual Tonsillitis 326

Hypertrophy of the Lingual Tonsil 326

Treatment 326

Hyperkeratosis 327

Treatment 327

Diseases of the Salivary Ducts 327

Stomatitis 327

Stenosis 327

Symptoms 328

Treatment 328

Diseases of the Salivary Glands 328

Secondary Stomatitis in Affections of the Salivary Ducts

Treatment 328

Diseases of the Salivary Glands in General Affections

Actinomycosis, Syphilis, Tuberculosis 328

Epidemic Parotitis 328

Treatment 328

Chronic Enlargement of the Salivary and Lacrimal Glands in Gen-

eral Disease 328

Treatment 328

Pyaralism 328

Opisthotonos 328

Phlegmons of the Buccal Cavity 328

Ludwig's Angina 328

Phlegmons of the Buccal Glands—	
Ludwig's Angina—	
Treatment	331
Afections of the Alveolar Processes	331
Parulis (Periostitis Alveolaris Dentalis)	331
Treatment	332
Pyorrhea Alveolaris	332
Treatment	332
Gingivitis	333
Afections of the Lips and Cheeks	333
Congenital Fistulae of the Lower Lip	333
Acute Cheilitis	334
Treatment	334
Chronic Cheilitis	334
Cheilitis Glandularis	334
Cheilitis Exfoliativa	334
Afections of the Malar Mucosa	335
Benign Tumors of the Mouth	335
Fibroma	335
Treatment	335
Lipoma	335
Treatment	336
Myxoma	336
Myoma	336
Chondroma, Osteoma	336
Hemangioma	336
Cavernous Angiomata	336
Treatment	336
Racemose Aneurysm	336
Lymphangioma	337
Diffuse Lymphoma	337
Treatment	337
Dermoid Cysts	337
Treatment	338
Cysts Originating from Glands	338
Treatment	338
Ranula	339
Treatment	339
Cysts at the Root of the Tongue	339
Struma of the Lingual Base	339
Treatment	340
Adenoma	340
Papilloma	340
Treatment	340
Endothelioma	340
Treatment	341
Malignant Tumors of the Mouth	341
Sarcoma	341
Treatment	342
Carcinoma	342
Carcinoma of the Lips	342
Treatment	342

Nervous Affections of the Mouth—**Paralysis of the Facial Nerve—**

Treatment 321

Glossodynia 321

Treatment 321

Vasomotor, Trophic and Secretory Disorders 321**Affections of the Tongue 322**

Malformation 322

Coating or Furring 322

Treatment 322

Lingua Geographica 323

Treatment 323

Hair-tongue (Lingua Nigra) 323

Treatment 323

Phlegmonous Processes of the Tongue 324

Abscess 324

Treatment 324

Acute Diffused Glossitis 324

Treatment 324

Decubital Ulcer of the Tongue 325

Treatment 325

Chronic Superficial Glossitis 325

Treatment 325

Acute Papular Glossitis 325

Treatment 326

Macroglossia 326

Lingua Plicata 326

Affections of the Lingual Tonsil 326

Acute Lingual Tonsillitis 326

Hypertrophy of the Lingual Tonsil 326

Treatment 326

Hyperkeratosis 327

Treatment 327

Diseases of the Salivary Ducts 327

Sialodochitis 327

Sialoliths 327

Symptoms 328

Treatment 328

Diseases of the Salivary Glands 328**Secondary Sialadenitis in Affections of the Salivary Ducts 328**

Treatment 328

Diseases of the Salivary Glands in General Affections 329

Actinomycosis, Syphilis, Tuberculosis 329

Epidemic Parotitis 329

Treatment 329

Chronic Enlargement of the Salivary and Lacrimal Glands (Mikulicz's Disease) 330

Treatment 330

Ptyalism 330

Aptyalism 330

Phlegmons of the Buccal Glands 330

Ludwig's Angina 330

Phlegmons of the Buccal Glands—**Ludwig's Angina—**

Treatment	331
---------------------	-----

Affections of the Alveolar Processes	331
--	-----

Parulis (Pericostitis Alveolaris Dentalis)	331
--	-----

Treatment	332
---------------------	-----

Pyorrhea Alveolaris	332
-------------------------------	-----

Treatment	332
---------------------	-----

Gingivitis	333
----------------------	-----

Affections of the Lips and Cheeks	333
---	-----

Congenital Fistulae of the Lower Lip	333
--	-----

Acute Cheilitis	334
---------------------------	-----

Treatment	334
---------------------	-----

Chronic Cheilitis	334
-----------------------------	-----

Cheilitis Glandularis	334
---------------------------------	-----

Cheilitis Exfoliativa	334
---------------------------------	-----

Affections of the Malar Mucosa	335
--	-----

Benign Tumors of the Mouth	335
--------------------------------------	-----

Fibroma	335
-------------------	-----

Treatment	335
---------------------	-----

Lipoma	335
------------------	-----

Treatment	336
---------------------	-----

Myxoma	336
------------------	-----

Myoma	336
-----------------	-----

Chondroma, Osteoma	336
------------------------------	-----

Hemangioma	336
----------------------	-----

Cavernous Angiomata	336
-------------------------------	-----

Treatment	336
---------------------	-----

Racemose Aneurysm	336
-----------------------------	-----

Lymphangioma	337
------------------------	-----

Diffuse Lymphoma	337
----------------------------	-----

Treatment	337
---------------------	-----

Dermoid Cysts	337
-------------------------	-----

Treatment	338
---------------------	-----

Cysts Originating from Glands	338
---	-----

Treatment	338
---------------------	-----

Ranula	339
------------------	-----

Treatment	339
---------------------	-----

Cysts at the Root of the Tongue	339
---	-----

Struma of the Lingual Base	339
--------------------------------------	-----

Treatment	340
---------------------	-----

Adenoma	340
-------------------	-----

Papilloma	340
---------------------	-----

Treatment	340
---------------------	-----

Endothelioma	340
------------------------	-----

Treatment	341
---------------------	-----

Malignant Tumors of the Mouth	341
---	-----

Sarcoma	341
-------------------	-----

Treatment	342
---------------------	-----

Carcinoma	342
---------------------	-----

Carcinoma of the Lips	342
---------------------------------	-----

Treatment	342
---------------------	-----

Malignant Tumors of the Mouth—

Carcinoma—	
Carcinoma of the Tongue	343
Treatment	344
Carcinoma of the Buccal Fundus	344
Carcinoma of the Malar Mucosa	344
Treatment	344
Carcinoma of the Palate	344
Treatment	344
Carcinoma of the Uvula	344
Tumors of the Maxilla	345
Fibroma	345
Chondroma	345
Osteoma	345
Odontoma	345
Cysts	345
Adamantoma	345
Sarcoma	346
Chondrosarcoma and Myxosarcoma	346
Carcinoma	346
Affections of the Pharynx	346
Acute and Chronic Pharyngitis	346
Treatment	347
Follicular Tonsillitis	347
Treatment	347
Parenchymatous Tonsillitis, Suppurative Tonsillitis, or Quinsy	347
Treatment	347
Chronic Tonsillitis	347
Hypertrophy of the Tonsils	347
Retropharyngeal Abscess	348
Treatment	348
Tuberculous Abscess	348

CHAPTER XVI.

DISEASES OF THE ESOPHAGUS.

Anatomy of the Esophagus	349
Deglutition Sounds	350
Instrumental Examination	350
Esophageal Bougie	350
Esophagoscope and Gastroscope	351
Inflammation of the Esophagus	353
Acute Esophagitis	353
Symptoms	353
Treatment	353
Chronic Esophagitis	353
Symptoms	354
Treatment	354
Esophageal Syringe (Rosenheim)	354
Exfoliative Esophagitis	355

Inflammation of the Esophagus—	
Exfoliative Esophagitis—	
Treatment	355
Fibrinous Esophagitis	355
Treatment	355
Phlegmonous Esophagitis	356
Treatment	356
Infectious Diseases in the Esophagus	356
Diphtheria	356
Variola	357
Skin Diseases in the Esophagus	357
Pemphigus and Herpes Zoster	357
Burns and Corrosions of the Esophagus	357
Treatment	358
Ulcers of the Esophagus	358
Gangrenous Ulcers	358
Decubital Ulcers	358
Tubercular Ulcers	359
Treatment	359
Syphilitic Ulcers	359
Diagnosis	360
Treatment	360
Peptic Ulcer	360
Symptoms	360
Treatment	360
Vegetable and Animal Parasites	361
Actinomycosis	361
Thrush	361
Animal Parasites	361
Structure of the Esophagus	361
Neoplasms in the Esophagus	362
Papilloma	362
Fibroma	362
Other Benign Neoplasms	362
Carcinoma	362
Symptoms	363
Diagnosis	363
Sarcoma	364
Cicatrical Stricture	364
Treatment of Esophageal Stricture	364
Technic of Introducing the Sound	365
Jaw-pointed Sound in Esophagus	365
Esophageal Sound (Crawcour,	367
Dilating the Stricture	368
Esophageal Dilators (Sippy, Senator, Schreiber)	370
Esophageal Bougie and Cannula (Leyden and Renvers)	371
Surgical Treatment	372
Alimentation	372
Symptomatic Treatment	373
Diverticula of the Esophagus	374
Traction Diverticula	374
Treatment	374

Diverticula of the Esophagus—	
Pubion Diverticula	374
Symptoms	375
Diagnosis	375
Prognosis	375
Treatment	376
Diverticular Sounds (Leube and Starck)	376
Foreign Bodies in the Esophagus	377
Other Causes of Esophageal Stricture	378
Thrush	378
Compression of the Esophagus	378
Spasm of the Esophagus	378
Symptoms	378
Treatment	379
Dilatation of the Esophagus	380
Etiology	380
Symptoms	380
Diagnosis	381
Prognosis	381
Treatment	381
Congenital Dilatation	382
Congenital Stricture	383
Rupture, Malacia, Perforation, Hemorrhage	383
Neuroses of the Esophagus	384
Hyperesthesia	384
Treatment	385
Anesthesia	385
Paralysis	385
Symptoms	385
Treatment	386
Atony	386
Treatment	386

CHAPTER XVII.

MOTOR NEUROSES.

<i>Vagotonia; Sympathicotonia; Hypermotility; Peristaltic Unrest; Cardiospasm; Pylorospasm; Eructations; Pneumatosis; Vomiting; Rumination; Regurgitation; Pyloric Insufficiency; Singultus Gastricus.</i>	
The Vegetative Nervous System	387
Vagotonia and Sympathicotonia	388
Diagnostic Phenomena in Disturbances of the Vegetative Nervous System	390
Aschner's Phenomenon	390
Hering's Phenomenon	390
The Pilocarpin Test	391
Differential Tables	391
Treatment	391
Hypermotility (Hyperkinesis)	392
Peristaltic Unrest of the Stomach	392
Treatment	392

CONTENTS

29

Cardiospasm	392
Symptoms	394
Diagnosis	394
Delineator String	394
Prognosis	395
Treatment	396
Oil Cure	397
Mechanical Treatment	397
Electrotherapy	398
Myer's Cardia Dilator	398
Surgical Treatment	398
Pylorospasm	398
Treatment	400
Technic of Dilating the Pylorus	400
Nervous Eructation (Aerophagy)	401
Diagnosis	402
Treatment	402
Pneumatosis (Drum-belly)	403
Treatment	403
Nervous Vomiting	403
Gastric Crises	403
Treatment	404
Rumination (Merycism)	405
Treatment	406
Regurgitation	406
Insufficiency of the Pylorus	407
Treatment	407
Singultus Gastricus (Hiccough)	408
Treatment	408

CHAPTER XVIII.

SENSORY NEUROSES.

Gastralgia; Hyperesthesia; Gastralgokenosis; Nausea; Bulimia; Akoria; Anorexia.

Gastralgia	410
Treatment	410
Gastric Hyperesthesia	413
Treatment	413
Gastralgokenosis (Stomach-ache)	414
Treatment	414
Nervous Nausea	415
Treatment	415
Bulimia	416
Treatment	416
Akoria	416
Treatment	416
Nervous Anorexia	416
Treatment	417

CHAPTER XIX.

NERVOUS DYSPEPSIA—NEURASTHENIA GASTRICA.

Etiology	418
Eye-strain	418
Cholecystitis, Constipation, etc.	419
Appendicitis	419
Symptoms	419
Prognosis	420
Prophylaxis	420
Treatment	421
Lactovegetable Diet	422
Physical Treatment	423
Sea-water Therapy	423
Apparatus for the Injection of Sea-water	424
Drug Treatment	425
Surgical Treatment	427
Umbilical Dyspepsia	428
Treatment	428

CHAPTER XX.

SECRETORY NEUROSES.

Hyperchlorhydria—Hyperacidity—Superacidity.

Etiology of Hyperchlorhydria	430
Pathology	431
Symptoms	431
Diagnosis	431
Prognosis	432
Treatment	432
Hygienic	432
Dietetic	432
Medicinal	435
Astringents	435
Atropin	435
Alkaloids	436
Hydrogen Peroxid	436
Analgesics	436
Acids	436
Alkalis	436
Course of Medication	437
Lavage of the Stomach	438

CHAPTER XXI.

SECRETORY NEUROSES (CONTINUED).

Hypersecretion—Gastrorrhea—Gastrosuccorrhea—Gastrochylorrhea.

Intermittent or Periodic Hypersecretion (Acute Intermittent Gastrorrhea)	440
Etiology	440

Intermittent or Periodic Hypersecretion (Acute Intermittent Gastrorrhea)	
Symptoms	440
Diagnosis	441
Treatment	441
Continuous Hypersecretion (Chronic Gastrorrhea)	441
Etology	442
Symptoms	442
Diagnosis	443
External Examination of the Stomach	443
Prognosis	443
Treatment	443
Diet	444
Medicinal Treatment	445
Lavage of the Stomach	446
Mineral Waters	447
Physical Treatment	447
Surgical Treatment	447
Alimentary Hypersecretion	447
Symptoms	447
Diagnosis	447
Treatment	448
Medicinal Treatment	448
Lavage of the Stomach	448

CHAPTER XXII.

ACUTE GASTRITIS: SIMPLE; INFECTIOUS; TOXIC; PHLEGMONOUS.

Simple Acute Gastritis	449
Etology	449
Pathology	449
Symptoms	450
Course	450
Prophylaxis	450
Treatment	450
Lavage	451
Emetics	451
Medication	451
Diet	452
Acute Infectious Gastritis	453
Etology	453
Pathology	453
Symptoms	454
Treatment	454
Toxic Gastritis	455
Etology	455
Pathology	455
Symptoms	456
Prognosis	456
Treatment	456
Phlegmonous Gastritis	457

Phlegmonous Gastritis—

Etiology	457
Pathology	458
Symptoms and Course	458
Treatment	459

CHAPTER XXIII.

CHRONIC GASTRITIS—ACID GASTRITIS—SUBACID GASTRITIS—ANACID
GASTRITIS; ACHYLIA GASTRICA.

Chronic Gastritis	460
Etiology	460
Pathology	461
Symptoms	461
Objective Symptoms	462
Diagnosis	462
Prognosis	463
Achylia Gastrica	464
Etiology	465
Pathology	465
Symptoms	465
Treatment of Chronic Gastritis and Achylia Gastrica	466
Diet	466
Medicinal Treatment	469
Hydrochloric Acid	469
Papain	469
Pancreatin	469
Stomachics	469
Treatment by Gastric Lavage	471
Treatment with Mineral Waters	472
Physical Treatment	472

CHAPTER XXIV.

MOTOR INSUFFICIENCY.

*Atony (Myasthenia); Dilatation (Ischachymia, Gastroclasis); Pyloric Stenosis;
Acute Dilatation of the Stomach.*

Motor Insufficiency of the First Degree (Atony)	473
Etiology	473
Symptoms	474
Diagnosis	474
Treatment	475
Diet in Normal Acidity, Hyperacidity and Hypersecretion	475
Diet in Subacidity and Anacidity	476
Lavage of the Stomach	476
Medicinal Treatment	477
Physical Treatment	477
Treatment with Mineral Waters	477

CONTENTS

33

Motor Insufficiency of the Second Degree (Dilatation)	478
Etiology	478
Symptoms	479
Diagnosis	480
Treatment	481
To Allay Thirst	481
Rectal Alimentation	482
Subcutaneous Nutrition	482
Treatment by Lavage	482
Mechanical Treatment	483
Physical Treatment	483
Mineral Waters	483
Medicinal Treatment	483
Treatment of Stenosis of the Pylorus	484
Dilatation	485
The Einhorn Pyloric Dilator	485
Surgical Treatment	485
Gastric Tetany	485
Acute Dilatation of the Stomach	486
Treatment	486

CHAPTER XXV.

GASTRIC ULCER.

Ulcus Ventriculi—Round Ulcer—Peptic Ulcer—Perforating Gastric Ulcer.

Pathology of Gastric Ulcer	488
Etiology	489
Frequency	489
Sex Predisposition and Age	489
Symptoms	490
Localization of Pain	491
Vomiting	491
Hemorrhage	491
Perforation	492
Appetite	492
Complications and Sequelæ	493
Diagnosis	493
Einhorn's "String Test"	493
Roentgenography in the Diagnosis	494
Prognosis	494
Treatment	495
Prophylaxis	495
Leube-Ziemssen Treatment	495
Lenhart's Treatment	498
Sippy Treatment	499
Einhorn's Duodenal Alimentation	500
Method of Procedure	501
Morgan's Modification	502
Medicinal Treatment	503
Alkaline Formulæ	503

Treatment—

Medicinal Treatment—	
Bismuth Salts	504
Silver Nitrate	505
Scarlet Red	505
Tincture of Iodin	506
Olive Oil	506
Treatment by Antilytic Serum	506
Treatment by Bacterial Vaccines	506
Surgical Intervention	507
Perforation in Gastric Ulcer	509
Subphrenic Abscess	509
Pyloric Stenosis	509
Hypertrophic Stenosis of the Pylorus	509
Rammstedt Operation	510

CHAPTER XXVI.

GASTRIC AND INTESTINAL HEMORRHAGE.

Diagnosis	511
Differential	512
Prophylaxis	512
Treatment	512
Lavage	513
Enemata	514
Hemostatics	514
Analgesics	518
Operative Treatment	520

CHAPTER XXVII.

EROSIONS; PERIGASTRITIS.

Erosions of the Stomach	521
Form	521
Etiology	521
Symptoms	522
Diagnosis	522
Pathology	522
Prognosis	523
Treatment	523
General Treatment	523
Local Treatment	523
Perigastritis	524
Hour-glass Contraction	524
Symptoms	525
Form	525
Diagnosis	525
Treatment	526

CHAPTER XXVIII.

ARTERIOSCLEROSIS; SYPHILIS; TUBERCULOSIS.

Arteriosclerosis	528
Etiology	528
Pathology	529
Symptoms	529
Diagnosis	530
Treatment	530
Syphilis of the Stomach	533
Diagnosis	533
Treatment	534
Tuberculosis of the Stomach	536
Forms	536
Treatment	536

CHAPTER XXIX.

TUMORS OF THE STOMACH.

Carcinoma; Sarcoma; Fibroma; Fibromyoma; Lipoma; Adenoma; Papilloma; Polypi; Hernia Epigastrica.

Carcinoma	537
Incidence	537
Etiology	537
Pathology	539
Forms	540
Medullary Carcinoma	540
Adenocarcinoma	540
Gelatinous or Colloid Carcinoma	540
Scirrhus Carcinoma	540
Ulcero-carcinoma	541
Complications	541
Symptoms	541
Diagnosis	542
Hemolytic Reactions	543
Antitryptic Reaction	544
The Mucostagmin Reaction	544
The Abderhalden Reaction	545
Blood-sugar Tolerance Test	544
Leukitis Plastica Hypertrophica	546
Treatment	546
Internal Treatment	546
Physical Treatment	549
Medicinal Treatment	549
Radiation Treatment	550
Treatment of Carcinoma of the Cardia	551
Sarcoma	553
Etiology	553
Pathology	553

Sarcoma—

Symptoms	553
Diagnosis	554
Treatment	554
Benign Tumors	555
Hernia Epigastrica	555

CHAPTER XXX.

GASTROENTEROPTOSIS.

*Gastropptosis; Enteroptosis; Splanchnoptosis; Coloptosis; Nephroptosis;
Hepatoptosis; Splenoptosis; Cecum Mobile; Redundant Sigmoid.*

Etiology of Gastroenteroptosis	557
Forms	557
Pathology	558
Habitus Enteroptoticus	558
Descent of Transverse Colon	560
Kinks of the Intestine	561
Symptoms	562
Diagnosis	564
Gastroenteroptosis	564
Nephroptosis	565
Palpation of Movable Kidney	567
Hepatoptosis	568
Splenoptosis	569
Prognosis of Gastroenteroptosis	569
Prophylaxis	569
Treatment	569
Hyperalimentation	569
Technic of Nutrition	571
Hydrotherapeutics	573
Massage and Exercise	574
Electrotherapeutics	574
Mechanical Treatment of Gastroenteroptosis	574
Bandages	575
Aaron's Abdominal Bandage	576
Adhesive Plaster Bandage	578
Corsets	578
Adhesive Belt	579
Mechanical Treatment	581
Surgical Treatment	583

CHAPTER XXXI.

DISEASES OF THE LIVER.

*Hepatitis, Cholecystitis, Gallstones, Ectopy, Fibrosis, Cirrhosis;
Ectopy, Syphilis, Tuberculosis, Neoplasms, Parasites, Fatty Liver;
Hepatoptosis, Neuropathy.*

Acute Affections of the Liver	584
Acute Inflammation of the Liver	584
Treatment	584

Acute Affections of the Liver—	
Abscess of the Liver	584
Symptoms	585
Treatment	586
Acute Yellow Atrophy of the Liver	586
Treatment	587
Febria Icterus	587
Treatment	588
Chronic Affections of the Liver	
Active Hyperemia	588
Diagnosis	589
Treatment	589
Passive Hyperemia	589
Symptoms	589
Treatment	590
Atrophic Cirrhosis	590
Etiology	590
Pathology	590
Symptoms	591
Tests for Lipase	592
Levulose Test	593
Phthalein Test	593
Urobilin	593
Prognosis	593
Treatment	594
General and Medicinal	594
Surgical	595
Hypertrophic Cirrhosis	596
Etiology	596
Symptoms	596
Diagnosis	597
Prognosis	597
Treatment	597
Hypertrophic Cirrhosis of the Liver in Bronze Diabetes	597
Treatment	597
Biliary Cirrhosis	597
Treatment	598
Cirrhosis Occurring in the Course of Affections of the Circulatory	
Organs	598
Atrophy of the Liver	598
Brown Atrophy	598
Red Atrophy	598
Partial Atrophy	598
Syphilis of the Liver	599
Congenital Syphilis	599
Acquired Syphilis	599
Symptoms	599
Diagnosis	600
Treatment	600
Tuberculosis of the Liver	600
Neoplasms of the Liver	600
Malignant Formations	600

Neoplasms of the Liver—

Malignant Formations—	
Carcinoma	600
Sarcoma	601
Treatment	602
Benign Neoplasms	602
Fibroma	602
Angioma	602
Cysts	602
Parasites of the Liver	602
Echinococci	602
Symptoms	603
Diagnosis	603
Treatment	604
Echinococcus Multilocularis	604
Other Parasites	604
Fatty Liver	604
Etiology	604
Pathology	605
Treatment	605
Hepatoptosis	605
Treatment	605
Neuralgia of the Liver	605

CHAPTER XXXII.

DISEASES OF THE BILE DUCTS AND GALL BLADDER.

Cholangitis; Cholecystitis; Catarrhal Jaundice; Hemorrhage; Neoplasms; Dilatation; Parasites; Gallstones.

Inflammation of the Bile Ducts and Gall Bladder	607
Simple Cholangitis and Cholecystitis	607
Pathology	607
Symptoms	608
Diagnosis	610
Hemocoetes	611
Treatment	611
Suppurative Cholangitis and Cholecystitis	613
Etiology	613
Pathology	613
Symptoms	614
Treatment	614
Hemorrhage into the Bile Ducts	614
Neoplasms of the Bile Ducts and Gall Bladder	614
Symptoms	615
Diagnosis	615
Treatment	615
Dilatation of the Biliary Organs	615
Hydrops and Empyema	616
Symptoms	616
Treatment	616
Parasites of the Bile Ducts	616
Diagnosis	616
Treatment	616

CONTENTS

39

Calculus	616
Symptoms	618
Diagnosis	618
Naunyn's Sign	619
Murphy's Sign	619
Examination of Duodenal Contents	619
Treatment	619
Anodynes and Narcotics	619
Diet	620
Medication	621
Non-surgical Biliary Drainage	621
Surgical Treatment	622

CHAPTER XXXIII.

DISEASES OF THE PANCREAS.

Pancreatitis; Achylia; Hemorrhage; Necrosis; Cysts; Tumors; Calculi.

Inflammation of the Pancreas	623
Chronic Pancreatitis	623
Etiology	624
Pathology	624
Diagnosis	624
Disturbance of Protein Digestion	624
Disturbance of Fat Digestion	625
Disturbance of Starch Digestion	625
Oil Test Breakfast	626
Loewi's Pupillary Reaction	626
Cambridge Reaction	626
Prognosis	627
Treatment	627
Regulation of Fats, Proteins and Carbohydrates	627
Surgical Treatment	628
Acute Pancreatitis	628
Treatment	629
Internal Treatment in Other Affections of the Pancreas	629
Hemorrhage of the Pancreas	631
Necrosis of the Pancreas	631
Pancreatic Cysts	632
Treatment	632
Tumors of the Pancreas	633
Treatment	633
Pancreatic Calculi	633
Treatment	633
Pancreatic Infantilism	634
Treatment	634

CHAPTER XXXIV.

ACUTE ENTEROCOLITIS.

*Acute Intestinal Catarrh—Acute Gastroenteritis—Acute Colitis—Cholera
Morbus—Cholera Nostras Acute Diarrhea.*

Etiology	635
Acute Infectious Catarrh	635

Etiology—	
Alimentary Catarrh	635
Catarrh Due to Exposure to Cold	635
Catarrh from Intoxication	635
Pathology	635
Symptoms	636
Diagnosis	637
Prognosis	638
Treatment	638
Treatment of Cholera Morbus	642

CHAPTER XXXV.

CHRONIC ENTEROCOLITIS.

Chronic Intestinal Catarrh—Chronic Enteritis—Chronic Colitis—Chronic Sigmoiditis.

Pathology	644
Symptoms	644
Diagnosis	645
Prognosis	647
Treatment	647
Of Cases with Diarrhea	647
Of Cases Associated with Constipation	650
Irrigation	651

CHAPTER XXXVI.

ENTERITIS MEMBRANACEA.

Mucosmembranous Enteritis—Mucous Colitis—Pseudomembranous Enteritis—Tubular Diarrhea.

Pathology	652
Symptoms	653
Treatment	653
Dietetic	654
Laxatives	656
Hydrotherapeutic Measures	656
Palliative Treatment	658

CHAPTER XXXVII.

CHRONIC CONSTIPATION.

Atonic Constipation, Spastic Constipation, Fragmentary Constipation.

Atonic Constipation	659
Symptoms	660
Treatment	661
Sandal and Bran	662
Agar	662
Liquid Petrolatum	664
Grape Cures	664
Massage	664
Lavage	666
Yeast	666

Atonic Constipation—	
Treatment—	
Hormonal	667
Surgical Treatment	668
Spastic Constipation	668
Diagnosis	669
Treatment	670
Dietetic	670
Mechanical	671
Electrical	671
Hot and Cold Air Douche	671
Enemata	671
Purgatives	672
Frangmentary Constipation	672
Treatment	672

CHAPTER XXXVIII.

CHRONIC DIARRHEA.

Gastrogenic Diarrhea; Intestinal Fermentative Dyspepsia; Nervous Diarrhea.

Gastrogenic Diarrhea	673
Symptoms	675
Treatment	676
Intestinal Fermentative Dyspepsia	677
Diagnosis	678
Treatment	679
Nervous Diarrhea	680
Symptoms	681
Diagnosis	681
Treatment	681

CHAPTER XXXIX.

INTESTINAL TOXEMIA, INTESTINAL STASIS, AND ILEAL REGURGITATION.

Intestinal Toxemia	683
Intestinal Stasis	683
Ileal Regurgitation	684
Etiology	684
Course of Intestinal Toxemia	685
Bacterial Growth	686
Indican	686
Types of Intestinal Putrefaction	687
Indolic Type	687
Saccharobutyric Type	687
Combined Indolic and Saccharobutyric Type	688
Symptoms	688
Treatment	689
Antiseptic Diet	689
Whey	690
Buttermilk	690
Sour Milk	690
Carbohydrates	690

Treatment—

Antagonistic Bacteria	691
Antiseptic Medication	692
Treating the Constipation	694
Duodenal Lavage	695
Mechanical Treatment	696
Surgical Treatment	696

CHAPTER XL.

FLATULENCE, METEORISM, AND TYMPANITES.

Origin of Cases	698
Exogenous	699
Endogenous	699
Microorganisms	699
Food Decomposition	700
Treatment	700
Diet	701
Massage	702
Medication	702

CHAPTER XLI.

ULCERS OF THE INTESTINE.

*Duodenal Ulcer—Ulcus Rotundum Duodeni—Peptic Ulcer of the Duodenum,
Jejunal Ulcer.*

Duodenal Ulcer	705
Etiology	705
Symptoms	706
Diagnosis	707
Palpation and Percussion	708
Determination of Gastric Secretion	709
The Test for Occult Blood	709
Roentgen Ray	710
Polycythemia	710
Complications	710
Prognosis	711
Treatment	711
Dietetic and Medicinal	711
Surgical	712
Jejunal Ulcer	712

CHAPTER XLII.

ULCERS OF THE INTESTINE (CONTINUED).

Typhoid Ulcers	713
Dietetic Treatment	713
High Caloric Diet	714
Caloric Table	715
Diet Table	717
Preparation of Vegetable Soup	717
General Directions for Feeding	"

CHAPTER XLIII.

ULCERS OF THE INTESTINE (CONTINUED).

Acute and Chronic Dysentery	719
Etiology	719
Pathology	720
Symptoms	720
Complications	721
Prophylaxis	721
Prognosis	721
Treatment of Acute Dysentery	722
Ipecacuanha	722
Emetin	723
Benzyl Benzoate as a Synergist	724
Emetin-Bismuth Iodid	724
Epinephrin	725
Arsphenamine	725
Bismuth Subnitrate	726
Antidysenteric Serum	727
Vaccine	727
Treatment of Chronic Dysentery	727
Medicinal	728
Surgical	729

CHAPTER XLIV.

ULCERS OF THE INTESTINE (CONTINUED).

*Catarrhal and Follicular Ulcers—Ulcerative Colitis—Ulcerative Enteritis—
Ulcerative Sigmoiditis; Stercoral or Decubital Ulcers.*

Catarrhal and Follicular Ulcers	730
Symptoms	731
Diagnosis	731
Treatment	732
Dietetic	732
Creosote and Cod-liver Oil	732
Lavage	733
Dry Treatment	733
Vaccines	735
Surgical Treatment	735
Stercoral or Decubital Ulcers	736

CHAPTER XLV.

Tuberculosis, Syphilis; Embolus, Thrombus.

Tubercular Intestinal Ulcers	737
Diagnosis	738
Prognosis	738
Treatment	739
Medicinal	739
Symptomatic	739
Specific	740

Tuberculosis of the Cecum	7
Symptoms	7
Treatment	7
Syphilitic Ulcers of the Intestine	7
Treatment	7
Embolie and Thrombotic Ulcers	7

CHAPTER XLVI.

OBSTRUCTION OF THE INTESTINE.

Ileus—Intestinal Occlusion—Miserere—Passio Iliaco.

Etiology	7
External Ileus	7
Internal Ileus	7
Paralytic and Spastic Ileus	7
Acute Flexure of the Sigmoid	7
Symptoms	7
Treatment	7
Surgical Treatment	7
Massage	7
Diet	7
Lavage	7
Enemata	7
Medication	7

CHAPTER XLVII.

STRICTURES OF THE INTESTINE.

Strictures of the Small Intestine	7
Symptoms	7
Strictures of the Large Intestine	7
Symptoms	7
Treatment of Intestinal Strictures	7
Medication	7

CHAPTER XLVIII.

TUMORS OF THE INTESTINE.

<i>Carcinoma; Sarcoma; Lymphosarcoma; Adenoma; Polypi; Lipoma; My</i>	
Malignant Neoplasms of the Intestine	7
Carcinoma	7
Pathology	7
Carcinoma of the Small Intestine	7
Symptoms	7
Diagnosis	7
Carcinoma of the Colon	7
Symptoms	7
Diagnosis	7
Treatment of Carcinoma of the Intestine	7
Sarcoma and Lymphosarcoma of the Intestine	7
Symptoms	7
Treatment	7
Benign Neoplasms of the Intestine	7

CHAPTER XLIX.

APPENDICITIS.

*periducular Inflammation—Circumscribed Peritonitis—Paratyphlitis—
Perityphlitis—Scolecciditis—Scolecutis.*

Appendicitis	767
ptoms	768
gnosis	769
McBurney's Sign	770
Meltzer's Sign	770
Blumberg's Sign	770
Blaisdell's Sign	770
Traction on the Spermatic Cord	770
Cecum Mobile	770
Appendicitis	771
ptoms	771
Appendicitis Larvata	772
gnosis	772
Rovsing-Chase Sign	772
Rutkevich's Adduction Sign	772
Bastedo's Dilatation Sign	773
Morris's Sign	773
Aaron's Sign	773
Friedman's Sign	773
nt of Acute Appendicitis	773
ly Operation	773
·Ochsner Method	774
gatives	776
t	777
rapha and Opium	778
nt of Chronic Appendicitis	779
aking Cures	779
unes and Phylacogens	780

CHAPTER L.

NERVOUS DISEASES OF THE INTESTINE.

nterospasm; Tormina Intestinorum Nervosa; Parens; Enteralgia.

psm	781
ptoms	781
atment	781
a Intestinorum Nervosa	782
atment	782
of the Intestine	782
atment	783
ga (Enteralgia Nervosa - Intestinal Colic—Colica Flatulenta)	783
atment	784

Tuberculosis of the Cecum	740
Symptoms	740
Treatment	741
Syphilitic Ulcers of the Intestine	741
Treatment	741
Embolio and Thrombotic Ulcers	741

CHAPTER XLVI.

OBSTRUCTION OF THE INTESTINE.

Ileus—Intestinal Occlusion—Miserere—Passio Iliaco.

Etiology	742
External Ileus	742
Internal Ileus	743
Paralytic and Spastic Ileus	744
Acute Flexure of the Sigmoid	745
Symptoms	745
Treatment	747
Surgical Treatment	747
Massage	749
Diet	749
Lavage	750
Enemata	751
Medication	753

CHAPTER XLVII.

STRICTURES OF THE INTESTINE.

Strictures of the Small Intestine	755
Symptoms	755
Strictures of the Large Intestine	757
Symptoms	757
Treatment of Intestinal Strictures	758
Medication	760

CHAPTER XLVIII.

TUMORS OF THE INTESTINE.

Carcinoma; Sarcoma; Lymphosarcoma; Adenoma; Polypi; Lipoma; Myoma.

Malignant Neoplasms of the Intestine	761
Carcinoma	761
Pathology	762
Carcinoma of the Small Intestine	762
Symptoms	763
Diagnosis	763
Carcinoma of the Colon	764
Symptoms	764
Diagnosis	764
Treatment of Carcinoma of the Intestine	765
Sarcoma and Lymphosarcoma of the Intestine	766
Symptoms	767
Treatment	767
Benign Neoplasms of the Intestine	767

CHAPTER XLIX.

APPENDICITIS.

*Appendicular Inflammation—Circumscribed Peritonitis—Paratyphlitis—
Perityphlitis—Scoleccoiditis—Scollecitis.*

Acute Appendicitis	767
Symptoms	768
Diagnosis	769
McBurney's Sign	770
Meltzer's Sign	770
Blumberg's Sign	770
Blausdell's Sign	770
Traction on the Spermatic Cord	770
Cecum Mobile	770
Chronic Appendicitis	771
Symptoms	771
Appendicitis Larvata	772
Diagnosis	772
Rovsing-Chase Sign	772
Rutkevich's Adduction Sign	772
Bastedo's Dilatation Sign	773
Morris's Sign	773
Aaron's Sign	773
Friedman's Sign	773
Treatment of Acute Appendicitis	773
Early Operation	773
The Ochsner Method	774
Purgatives	776
Diet	777
Morphin and Opium	778
Treatment of Chronic Appendicitis	779
Drinking Cures	779
Vaccines and Phylacogens	780

CHAPTER L.

NERVOUS DISEASES OF THE INTESTINE.

Enterospasm; Termina Intestinorum Nervosa; Paresis; Enteralgia.

Enterospasm	781
Symptoms	781
Treatment	781
Termina Intestinorum Nervosa	782
Treatment	782
Paresis of the Intestine	782
Treatment	783
Enteralgia (Enteralgia Nervosa—Intestinal Colic—Colica Flatulenta)	783
Treatment	784

Proctitis	841
Symptoms	841
Diagnosis	841
Treatment	842
Ulcers of the Rectum	843
Diagnosis	844
Treatment	844
Prolapse of the Rectum (Procidentia Recti)	845
Treatment	846
Rectal Truss (Esmarch)	847
Proctospasm	848
Treatment	849
Atzperger's Refrigerator	849
Paresis and Paralysis of the Rectum	849
Treatment	849
Coccygodynia	850
Treatment	850

CHAPTER LIV.

DISEASES OF THE ANUS.

Pruritus Ani; Anal Fistula; Fissure of the Anus.

Pruritus Ani	851
Pathogenesis	851
Local and General Conditions	852
Treatment	853
General	853
Local	853
Medicinal	854
Dilatation	855
Roentgen Ray	856
Bacterial Vaccines	856
Surgical Measures	857
Anal Fistula	857
Diagnosis	858
Treatment	858
Fissure of the Anus	858
Symptoms	859
Treatment	859
General	860
Local	860
Cauterization	860
Baths	861
Pessaries	861
Electricity	861
Divulsion of the Anal Sphincters	861
Surgical Treatment	862
Comparative Scales of the Metric and Ordinary Weights and Measures	862
Index	863

DISEASES OF THE DIGESTIVE ORGANS.

CHAPTER I.

THE PHYSIOLOGY OF DIGESTION.

THE physiology of digestion appeals to the physician, the physiologist, and the chemist from slightly varying viewpoints. To the physiologist and the chemist the process itself is the chief concern. The clinician must go farther: he must not only be conversant with the changes which take place under normal conditions, but he must be able to make the necessary deductions when called upon to treat abnormal digestion. While the physiologist and the chemist study the stomach or the action of the gastric secretion, the physician has to consider this organ in its relation to oral and intestinal digestion also.

Digestion proper begins with the mastication and insalivation of food. The food becomes more or less intimately incorporated with the saliva before being swallowed, and, as we shall see, the process begun in the mouth continues in the stomach. It is important, therefore, that the condition of the mouth should be the best possible. Putrefactive processes, if present, should receive prompt attention, and the dentist should be consulted at regular intervals.

By the term *digestion* is understood the process of rendering food material absorbable, a process which is accomplished by the disintegrating and dissolving action of secretions containing enzymes, assisted to a greater or less extent by mechanical action. These ferments or enzymes are found in the saliva, gastric juice, bile, and pancreatic and intestinal secretions.

SALIVARY DIGESTION.

Action of the Saliva. In man and in most of the higher animals the saliva has a twofold action—physical and chemical. The physical action of saliva consists in the moistening of the food, facilitating mastication by the teeth; moreover, by virtue of the mucus it contains, all the passages become lubricated, rendering more easy the act of deglutition and the passage of the bolus of food into the stomach. In dogs the physical action of saliva is the only one. In herbivorous and in omnivorous animals, including

man, the saliva has a chemical action also, which is very important in its relation to the digestion of starch. Saliva has a specific gravity of 1.002. The secretion from the parotid gland contains a ferment, ptyalin, which possesses the property of converting starch into dextrin or maltose. While the action of the amylase, ptyalin, begins with the food in the mouth, the greater portion of salivary digestion is performed during the first period of digestion in the stomach; for though the partaking of food causes almost immediate secretion of hydrochloric acid by the gastric glands, some time must elapse (from twenty to forty minutes) before the acid secretion of the stomach can penetrate the food sufficiently to inhibit salivary digestion. The food material at the fundus of the stomach may remain undisturbed for a considerable time and thus escape mixture with the acid gastric juice. Complete mastication of food, in order that the saliva may become thoroughly incorporated with it, is imperative for complete amylolysis.

Ptyalin.—Ptyalin, or the diastatic ferment of the saliva, converts starches as well as glycogen into sugar. This ferment acts in a slightly alkaline or neutral medium. Starches are first converted into maltose or isomaltose, from which dextrose appears to be a result of inversion by maltase. The change takes place to better advantage in cooked than in raw starch. The several intermediate stages in the transformation of starches are as follows: The starch becomes liquefied so that it forms a true solution rather than a suspension. The product of the initial stage of salivary digestion is known as amylopectin, and turns blue when treated with a dilute Lugol solution. As the process continues, the color produced by the Lugol solution gradually changes from a blue to a violet red and finally to a mahogany brown. Starches modified to this extent are known as erythropectin. As the process of salivary digestion continues still further, no color change is obtained from the addition of the Lugol solution; the term achropectin is used to designate the product of this stage of the digestive process. These changes may be summarized as follows:

- | | |
|--|---|
| 1. Amylopectin (amylulin, soluble starches). | Stains blue with iodine or Lugol solution. |
| 2. Erythropectin. | Lugol solution changes first to a violet blue, then red violet, and finally mahogany brown. |
| 3. Achropectin. | No color change produced by Lugol solution. |
| 4. Maltose. | |
| 5. Dextrose. | |

MOVEMENTS OF THE STOMACH.

Solid food remains in the stomach for several hours, where it is subjected to the action of a special fluid, the gastric juice. During this time, by muscular contractions of the walls of the stomach, the thinner portions of the chymified material are ejected through

the pylorus into the intestine. The tonic closure of the sphincters at the cardia and pylorus shuts off the food from the remainder of the alimentary canal except at such times as there is a relaxation of the pylorus to permit the passage of chyme into the duodenum. During the initial stages of gastric digestion the pylorus is closed so firmly that upon excision of the stomach none of its contents will escape. As digestion advances, however, the pylorus offers less and less resistance, until finally it yields to permit the passage into the duodenum of digested gastric contents.

Since the discovery of the Roentgen ray, interesting studies have been made of the movements of the stomach. Cannon, among others, has devoted much attention to the subject. By giving an animal food mixed with bismuth subnitrate, he was able to obtain roentgenograms of the stomach, the bismuth being opaque to the Roentgen ray. From these studies it has been confirmed that peristaltic movements take place soon after the entrance of food into the stomach. The stomach "consists of two parts physiologically distinct" the cardiac portion, a food reservoir in which salivary digestion continues, and the pyloric portion, the seat of active gastric digestion. The food passes from the former to the latter by tonic contraction of the muscles.

The peristaltic muscular activity is confined to the pyloric portion. The efficiency of peristalsis in mixing the food depends upon the contraction of the pyloric sphincter, so that each peristaltic ring or contraction wave forces the gastric contents into a blind pouch. Unable to pass out through the pyloric exit, the food is forced back through a succeeding peristaltic ring, and in this way is brought thoroughly under the influence of the glandular secretions of the pyloric portion of the stomach. In the human stomach during digestion the peristaltic waves occur at intervals of about twenty seconds. In periods of relaxation of the pyloric sphincter, as digestion progresses, these contraction waves force some of the fluid contents of the stomach into the duodenum. After the propulsion of a certain quantity of fluid chyme into the intestine the pylorus remains closed until the acid on the distal side of the pyloric sphincter becomes neutralized by the bile and the alkaline pancreatic secretion in the duodenum. The acid chyme provides a chemical stimulus for pancreatic secretion. The opening and closing of the pylorus is also dependent upon food being swallowed with an appetite. We now know there is a psychic motility of the stomach similar to its psychic secretion.

By mixing bismuth subnitrate with the food and obtaining a roentgenogram of the stomach during the process of digestion, it has been learned further that carbohydrate foods begin to pass out of the stomach in a comparatively short time after ingestion, requiring only about one-half as much time for gastric digestion as proteins. When taken alone, fats have been found to remain for

a long time in the stomach, and when taken along with other foods they delay to a marked extent the passage of the whole chymified food mass into the intestine. If carbohydrates be fed before proteins in an experimental diet, the former, being nearest the pyloric portion of the stomach, will be almost immediately propelled into the intestinal canal, leaving the protein behind to be acted upon by the gastric juice. To reverse the order of feeding will retard the passage of carbohydrates into the duodenum.

Liquids pass through the empty stomach by a well defined route along the small curvature called the "water way" of the stomach. Even when the stomach is full, some of the liquids follow this path and directly enter the duodenum.

The stomach is essentially an automatic organ. The excised stomach when kept at the temperature of the body continues to execute regular movements. It has nerve plexuses within its walls and is also connected with the cerebrospinal and sympathetic systems. During digestion the normal peristaltic movements of the stomach are in all probability due to a local reflex from Auerbach's plexus. Stimulation of the sympathetic fibers has an inhibitory effect upon gastric peristalsis. It has been found that the impulses received along the path of the vagus are motor. The automatic rhythmical contraction is inherent in the muscular coat of the stomach, however, and is merely regulated by impulses from the central nervous system passing down the vagi, and from the sympathetic system by way of the splanchnic nerves. The pyloric sphincter as well as the remainder of the musculature of the stomach is supplied by motor fibers from the vagus; on the other hand, stimulation of the splanchnic nerves causes the contracted stomach to dilate and the pylorus to relax.

GASTRIC DIGESTION.

We are indebted to Pawlow, the Russian investigator, for new knowledge concerning the physiology of digestion, especially that portion of the subject which is most directly concerned with gastric and duodenal secretion. Pawlow's experiments enabled him to study the gastric secretion in dogs after feeding certain foods, and the effect of the so-called sham feeding upon gastric secretion. This investigator has also studied the relation between the action of gastric and that of pancreatic juice. These studies were facilitated by the establishment of a gastric fistula leading from a blind pouch or cul-de-sac. We have learned that the glands of the stomach continue to secrete gastric juice until the food enters the duodenum, the quantity of secretion being in proportion to the quality of food ingested. While the secretion of the stomach under normal conditions is always acid, the acidity increases as the gastric juice is more rapidly secreted. Furthermore, the digestive

power of the gastric juice is subject to variation, depending upon the kind of food ingested. Gastric juice secreted after a bread diet is said to possess the greatest digestive power, while that of least strength follows the partaking of a purely milk diet. The total acidity, on the other hand, is greatest after meat and lowest after bread diet. From the point of view of weight, meat requires the greatest and milk the smallest amount of gastric juice. In the majority of cases the so-called psychic secretion, or that produced by the sight, taste, or odor of food, constitutes the commencement of gastric secretion. Such substances as meat broths and meat juices or solutions of meat extracts are excellent stimulants to gastric secretion. After gastric secretion has begun, further digestive power is developed by the ingestion of bread and egg foods. The amalgamation of protein and starch in bread accounts for the high digestive power that "bread juice" is said to contain. Fats have the effect of diminishing or inhibiting secretion; they do not in any way stimulate it.

Enzymes.—The study of enzymes has engrossed the attention of a number of observers during recent years. The commonly accepted view of the mode of action of these ferments is that originally propounded by Ostwald, namely, that they act by catalysis. The term is employed by chemists to designate a kind of reaction which is brought about by the mere contact or presence of certain substances known as catalyzers, which themselves appear to remain unchanged. As defined by Stirling, a catalyzer is a substance which will increase the velocity of a reaction without adding in any way to the energy changes involved in the reaction or taking part in the formation of end products. The activity of enzymes appears to be specific in character; *e. g.*, those ferments which act upon carbohydrates are not capable of producing any effect upon fats or proteins.

The enzymes of the body are colloidal in structure, with an unknown composition. Most of them are soluble in water, glycerin, or physiologic salt solution. They are destroyed completely by high temperatures (140° to 175° F.), and their physiologic action is retarded in whole or in part by temperatures only slightly below the normal. The enzymes are capable of their greatest activity at the temperature of the human body. They may be precipitated from solution, in part at least, by alcohol, which property is utilized in obtaining purified specimens. Enzymes may exist in an inactive or latent form in the cells which produce them, and may be still inactive after they are secreted. The inactive or latent forms of enzymes are known as zymogens or proenzymes. Before the zymogen can become effectual, it requires the aid of some other agent. The inorganic substances which render enzymes active agents in digestion are known as activators; organic substances which produce the same result are called *kinases*.

The fundus and the pyloric portion of the stomach are supplied with tubular glands which exhibit marked differences in structure in the two parts. In man the tubular glands of the fundus are provided with a duct lined with simple columnar epithelial cells, into which duct empty one or two secreting tubules supplied with two varieties of epithelial cells, namely, central or peptic cells, and parietal or oxyntic cells. In the pyloric portion of the stomach there is only the one kind of cell, namely, the peptic. The parietal or oxyntic cells secrete acid, while the central or peptic cells provide the pepsinogen or pepsin and rennin for gastric digestion.

Pepsin.—Pepsin, or rather pepsinogen, is active only in the presence of free hydrochloric acid. Hydrochloric acid possesses the property of converting pepsinogen into pepsin more thoroughly than can be done by any other mineral acid. Pepsinogen, or pepsin in the latent state, has such a high resistant power that it is present even in markedly advanced stages of catarrhal gastritis, as well as in carcinoma. Peptids and albumoses constitute the end-result of peptic digestion. The conversion of proteins and gelatinous substances into soluble albumoses takes place by degrees, so gradually in fact that it is difficult to determine the intermediate products of the process.

Hydrochloric Acid.—Hydrochloric acid acts in various ways in performing and facilitating the normal process of digestion. In the first place, it is antizymotic and antiseptic, destroying pathogenic microorganisms and arresting fermentation and putrefaction; the antiseptic action of hydrochloric acid continues in the duodenum. It also acts as a means of regulating peristalsis. Hydrochloric acid with pepsin converts food proteins into albumoses; pepsin, however, is the chief agent in this transformation process, hydrochloric acid acting as an adjuvant. By hydrochloric acid cane-sugar is converted into dextrose and levulose.

Normal Gastric Juice.—Normal gastric juice is a thin, colorless or nearly colorless fluid, with a strongly acid reaction and a characteristic odor; its specific gravity is about 1.002. The acidity of the gastric juice is due to the presence of free hydrochloric acid, the amount of which varies according to the duration of digestion. The acidity at the beginning of digestion is low, owing to the fact that a portion of the acid is neutralized by the alkalinity of the saliva incorporated with the food. While the gastric juice has a more or less constant acidity, its reaction may be diminished by alkalis in the stomach, or by combination with the protein of the food, forming acid-albumins or syntonins. The normal acidity of the gastric juice of man, estimated to be 0.2 per cent., may, according to Hornberg, reach 0.4 or 0.5 per cent. during digestion.

Pawlow, in his work on the digestive glands, has demonstrated that gastric secretion is under the control of the nervous system, and that the secretory fibers are contained in the vagus. If the

vagus be cut below the origin of the recurrent laryngeal, so as to avoid paralysis of the larynx, and sham feeding performed, there is no gastric secretion—proving conclusively that the vagus contains the secretory fibers. The hypothesis is confirmed by stimulation of the peripheral end of the cut nerve. Pawlow's experiment, which consists in dividing the esophagus of a dog in the neck, and connecting the esophageal mucous membrane with the skin to form a fistulous opening, is well known. Food fed to Pawlow's dogs escaped through the fistulous opening in the esophagus without reaching the stomach. The sham meal, as the experimenter designates it, had the effect of producing a copious flow of gastric juice, so long as the vagus was intact. The flow of gastric juice resulted evidently from a stimulation of the secretory fibers of the vagus, by the sensations of sight, odor, taste, etc., during the masticating and swallowing of food. The beginning of gastric secretion is psychic.

Under normal conditions gastric juice continues to be secreted as long as food remains in the stomach. Mechanical stimulation of the gastric mucous membrane has no effect upon the secretion of the glands of the stomach. The sensation of eating serves to start the secretion in an ordinary meal. The afferent stimuli originate in the mouth and nostrils and, as stated, probably with the sense of sight. The efferent path is through the vagus. Some food articles, among which are meat extracts, meat juices, and soups, and in a less degree milk and water, are said to contain substances which, when taken into the stomach, promote gastric secretion.

Gastrin.—Decoctions of the mucous membrane of the pylorus injected into the blood are found to increase the secretion of gastric juice. According to Elkins, secretagogues preformed in the food are produced during digestion act upon the mucous membrane of the pylorus, giving rise to a "gastrin," or gastric "secretin," which, after absorption into the blood, is carried to the gastric glands and stimulates them to secretion. These chemical messengers determining the various secretions, such as gastric, pancreatic, biliary, and intestinal, have been designated *hormones* (from ὁρμῶν, to arouse or excite) (Starling).

Pepsin. The principal action of pepsin consists in the conversion of the proteins of the food into peptones. Soluble protein, after passing through several intermediate stages, the results of which have been isolated and named acid-albumin, parapeptone, and peptone, becomes peptone. The first step in the digestion of protein consists in its conversion into an acid-albumin (syntonin). Under the action of pepsin, syntonin or acid-albumin undergoes hydrolysis, producing protalbumoses. Under the continued influence of pepsin these bodies undergo further hydrolysis, with the consequent formation of secondary proteoses (deutero-albumoses).

The further hydrolysis of the secondary proteoses results in the production of peptones.

Rennin.—Rennin is analogous to pepsin in that it is formed in the peptic or central cells and is present in the cells as a zymogen. The conversion of prorennin into the active enzyme takes place very readily under the influence of hydrochloric acid. Rennin curdles the casein of milk, and this apparently is its only action in the stomach. Casein, the chief protein in milk, has an important nutritive value. It is digested, like other proteins, by pepsin in the stomach and trypsin in the intestine, the end-result of the process of gastric digestion being peptone.

Lipase.—It has been demonstrated that the normal gastric mucosa in man secretes a lipase, or fat-splitting ferment, which acts readily upon the emulsified fats of milk, cream, or yolk of egg. This ferment, which is secreted by the cells of the fundus of the stomach, has been extracted by means of glycerin. It is inactive in an alkaline medium.

Fats in gastric digestion become liquefied by the heat of the body and, being thus set free from their intimate admixture with other foodstuffs, are distributed throughout the chyme by the movements of the stomach. In this way they are prepared for digestion by the pancreatic juice and bile in the intestine.

Absorptive Power of the Stomach.—It is probable that the absorptive power of the stomach is limited to such substances as salts, sugars, and dextrans that may have been formed from starch in salivary digestion. Absorption does not take place readily in the stomach; it is a distinctive feature of intestinal digestion. Water when taken alone is practically not at all absorbed from the stomach, but as soon as introduced begins to pass into the intestine in a series of spurts, by the contraction of the walls of the stomach.

INTESTINAL DIGESTION.

Digestion by the Small Intestine.—While the food remains in the stomach it undergoes a merely preparatory treatment that renders it suitable for intestinal digestion. The principal work in the digestive process is performed by the small intestine with the coöperation of the glands connected with it—the liver and the pancreas.

According to Pawlow's investigations it must be assumed that the intestinal mucosa is provided with organs of perception which are affected by the chemical quality of the intestinal contents, and which regulate by way of reflexes the work of the digestive glands and the mechanical function of the intestine. These reflexes are partly chemical, inducing the biliary flow and the secretion of pancreatic juice. In this process the quantity and composition of these juices adapt themselves closely to the composition of the

ingested food. Obviously, therefore, the digestive glands are highly secretory, as they must needs be in order to perform their allotted task.

Aside from the chemical reflexes, there are also motor reflexes at work, one of which belongs to the duodenal mucosa. The gastric hydrochloric acid acting on the intestinal mucosa regulates the opening and closing of the pylorus. As soon as the bile, pancreatic juice and succus entericus in the duodenum are acidified by the acid chyme, and hydrochloric acid is in contact with the duodenal mucosa, the pylorus closes itself. This acid also contracts a part of the duodenum adjacent to the pylorus.

Pancreatic Juice. The pancreas is the most important of the digestive glands. In the hungry state and during the period of digestive rest it is pale, relaxed, and inactive, but the moment food is introduced into the stomach it at once takes up its work, and, owing to increased blood supply, becomes distended and assumes a rose-red color. It begins to secrete juice as soon as the acid gastric contents enter the small intestine. The principal inciting factor of pancreatic secretion is the hydrochloric acid of the stomach as it enters the duodenum. The duodenal mucosa contains *prosecretin*, which is transformed into *secretin* by the hydrochloric acid. Secretin is absorbed and stimulates the flow of pancreatic juice. The pancreatic secretion is proportionate in quantity and alkalescence to the quantity and acidity of the gastric juice. The quantity of organic constituents it contains (ferments) is very small, while the quantity of inorganic constituents (alkalis) is exceedingly large. However, the juice of the pancreas is never quite devoid of ferments. The combination of the gastric juice with the pancreatic juice explains why the most important digestive processes can take place without any noteworthy change in the alkalinity of the blood. The acid component of the sodium chlorid contained in the blood (chlorin) enters the peptic glands and then the stomach, while the basic element (sodium) serves the pancreas as sodium carbonate in the preparation of its secretion. The two components of sodium chlorid become reunited as the acidity of the chyme is neutralized by the alkalinity of the pancreatic secretion.

Water, fats and psychic influences also stimulate the pancreatic secretion, while solutions of potassium or sodium bicarbonate have an inhibiting effect upon the formation of pancreatic juice.

The pancreatic solution is a limpid, colorless fluid, containing 0.2 to 0.4 per cent. of sodium carbonate and three ferments. The quantity secreted in twenty-four hours depends upon the quantity and composition of the ingested food.

The three ferments of the pancreatic secretion are:

1. *Trypsin*.—Trypsin in its pure state is found in the pancreatic juice after a pure meat diet; otherwise there will be found tryp-

sinogen, which is a proenzyme that is later activated into trypsin by the *enterokinase* of the intestinal juice. Enterokinase is only secreted when trypsinogen enters the intestine—so trypsinogen produces its own kinase by a specific chemoreflex. Peptic digestion comes to an end in the stomach when the proteins have been changed into albumoses, but trypsin continues the process by digesting the albumoses, thereby forming the foundation of the albuminous molecules or aminoacids.

2. *Amylopsin*.—Amylopsin seems to be present in the pancreatic juice as a complete ferment which changes starch into dextrin and maltose.

3. *Steapsin*.—Steapsin is the fat-splitting ferment of the pancreas, splitting neutral fats into glycerin and fatty acids, principally palmitic, stearic, and oleic. Like trypsinogen, the fat-splitting ferment must first be activated, and this is done by the bile.

The Bile. The quantity of bile secreted in the liver of an adult in one day amounts to from 400 to 800 Cc.—a pint to a pint and a half. Bile differs from the other digestive juices in the fact that it is continuously produced, even in hunger, and its composition is only slightly affected by the nature of the food. The bile thus continuously secreted is stored up in the gall bladder and is of a mucoviscous consistence owing to its becoming mixed on its way through the bile ducts and in the gall bladder with the muco-albuminous secretion of the gall-bladder mucosa. While in the gall bladder the bile becomes considerably inspissated from the loss of water and grows darker in color and more viscid.

The characteristic constituents of bile are the biliary acids and pigments. Other constituents are fats, soaps, lipoids (cholesterol, lecithin), nucleoprotein, urea, and mineral salts (salts of sodium, phosphorus, calcium, and magnesium).

The *biliary acids*, glycocholic and taurocholic, are found in the bile as sodium salts. Sodium glycocholate takes up water in the intestine under the influence of dilute acids and alkalis, and through the action of ferments is decomposed into glycochol (amino-acetic acid) and cholic acid. Sodium taurocholate is simply decomposed into taurin (amino-ethyl-sulphonic acid) and cholic acid.

The *biliary pigments* are bilirubin and biliverdin; they are non-ferrie derivatives of the blood pigment. Biliverdin is produced by oxidation of bilirubin. These pigments act like acids, forming soluble combinations with alkalis. In the intestine bilirubin is reduced to hydrobilirubin, the normal fecal pigment, through the action of intestinal bacteria.

The flow of bile into the intestine is controlled by a reflex mechanism dependent upon two constituents of the intestinal contents; peptone and fat. The entrance of peptone and fat into the duodenum causes the normal flow of bile into the intestine.

Bile does not seem to have any independent digestive power

of its own. It acts as an auxiliary to the activity of the pancreatic juice by activating, as stated above, the steapsin. Like the intestinal and pancreatic secretions, it emulsifies fats, owing to their containing easily decomposable alkalis. Its alkaline character also assists in neutralizing the acidity of the chyme coming from the stomach. Otherwise, bile can only be regarded as a secretion which carries into the intestine many metabolic end-products—for instance *cholesterol*, which is kept in solution in the presence of biliary acid salts. Cholesterol is probably a decomposition product which is regularly formed in the metabolic processes of the living protoplasm; it is demonstrable in many cells. The biliary acid salts are absorbed from the intestine and carried back to the liver.

The Intestinal Juice.—The consistence of intestinal juice differs according to location. In the upper segments of the intestine it is more liquid and watery, in the lower more viscous and mucous. These variations correspond to the different functions of the parts. In the small intestine the strongly acid chyme emerging from the stomach requires an abundant quantity of liquid alkaline juice to neutralize its acidity and to render the food constituents alkaline. In the large intestine, especially in its lower parts, a mucous consistence of the intestinal juice facilitates the passage of the feces and protects the intestinal mucosa from mechanical and chemical injury.

The juice of the small intestine is derived principally from the glands of Lieberkühn. Unlike other digestive juices, it is secreted through the agency of a local mechanical irritation. It contains 0.1 to 0.5 per cent. of calcium carbonate, 0.5 to 0.6 per cent. of sodium chlorid, and 5 per cent. of a mucoid albuminous substance which is regarded as a mucin or nucleoprotein.

Its high percentage of calcium carbonate indicates that its principal task is the neutralization of acids and the emulsification of fats. The acidity of the chyme is effectively neutralized by reaction, and with this process an important mechanical effect is associated. Just as, in the test tube, the intestinal juice becomes strongly effervescent upon addition of hydrochloric acid, so will food particles saturated with hydrochloric acid effervesce when coming in contact with the alkaline intestinal juice of the small intestine. As a consequence carbon dioxid is rapidly and freely developed, causing the smallest food particles to be disintegrated. This results in a loosening of the whole mass of chyme, so that the digestive ferments have free access to all its parts.

The juice of the small intestine contains a number of important ferments secreted by the epithelia of the tract. They are: maltase, lactase, invertin, erepsin, enterokinase, and probably also a cytase.

Maltase changes maltose into dextrose; lactase splits sugar of milk into dextrose and galactose; invertin splits cane-sugar into

dextrose and levulose. Erepsin does not attack intact protein, but decomposes its digestive products (albumoses and peptone) into aminoacids; casein is likewise split by erepsin. As already mentioned, enterokinase converts trypsinogen of the pancreatic juice into trypsin; it is not always present in the intestinal juice, for on mechanical irritation of the intestinal mucosa a juice is secreted which is free from enterokinase. Possibly enterokinase is related to the lymphatic apparatus of the intestinal mucosa. Investigations made by Lohrlich on cellulose digestion have made it appear probable that in human intestinal juice there is also a cellulose-dissolving ferment, or a cytase.

Absorption in the Small Intestine.—Absorption generally takes place through the numerous intestinal villi which are immersed in the chyle. These villi absorb water, salts, and all such substances as have become liquefied through the processes of digestion and are required by the organism to sustain its equilibrium and growth. The villi are thick and long in the upper segment of the small intestine, while in the ileum they are smaller and less dense.

Intestinal absorption is a function of the epithelia, and is not effected by osmosis. The gaseous or dissolved food constituents which have become absorbable are diffused in the epithelia of the intestinal mucosa and enter into chemical interrelation with their protoplasm. The protoplasm itself undergoes continuous disintegration and reintegration, demanding a supply of new material to take the place of that which is excreted into the blood or lymph vessels or into the intestinal lumen. Absorption continues through the capillaries and lacteals. The material in the capillaries is carried to the portal vein, which subdivides into capillaries in the liver, the capillaries becoming reunited as hepatic veins, anastomosing with the inferior vena cava. The lacteals go to the thoracic duct, which empties into the left subclavian vein.

Absorption of Protein. Man and all animals have the faculty of rebuilding protein from the aminoacids formed through the action of trypsin and erepsin. Of course every animal species has its own specific protein. Foreign protein, absorbed in its original form, always acts as a blood poison; from which it follows that it is necessary for the intestine to decompose the protein substances of a given food into simple and assimilable forms. The fundamental form is that of aminoacids. These aminoacids are absorbed from the small intestine and circulate in the blood as such. Practically all the nitrogen derived from ingested and digested proteins is found in the portal stream in the form of ultimate aminoacids. Absorption of undigested protein does not normally occur to any noteworthy extent.

Fat Absorption.—Absorption of fats can only occur after the fats have been converted into liquid form. Having been emulsified, their surface is so considerably extended that the fat-splitting

ferments can effectively act upon the small fat droplets. In this process, neutral fat is split into glycerin and fatty acids, and the latter are again changed through the alkali of the digestive juices into soluble, easily absorbable fat soaps. Simultaneously the glycerin is absorbed and enters directly into the absorptive epithelial cells, combining with the fatty acids set free from the soaps to form neutral fats, which can be observed in the deeper parts of the intestinal epithelia in the form of minute droplets. The fat droplets emerge from the epithelia and pass into the lacteals and then to the thoracic duct. Only the fats required for further use pass through the thoracic duct; they enter the blood stream and are either stored away as adipose or oxidized in heat production.

Carbohydrate Absorption.—Dextrose, levulose and galactose are absorbed as such, while glucose and sugar of milk are first converted into invertose. The absorption of dextrose and maltose takes place from hypertonic as well as hypotonic solutions. The utilization of starch occurs more slowly than that of pure sugar, because starch must first be converted into sugar. Those starchy articles of food which contain the least cellulose are most easily digested.

Absorption of Cellulose and Hemicellulose. Cellulose and hemicellulose are polysaccharids. Cellulose is an anhydrid of dextrose, while hemicelluloses are constituents of vegetable cell-walls which belong neither to starch nor to cellulose, and can be hydrolyzed with dilute mineral acids. Hexosane (galactan) and pentosane (arabin, xylan) are the principal hemicelluloses, and their sugars are galactose, arabinose, and xylose. As Lohrlich has demonstrated, both cellulose and hemicellulose are digestible by man, probably through a cytase of the intestinal juice. The normal intestine digests about 50 per cent. of the ingested quantity of cellulose and even a larger percentage of hemicellulose. This is shown by the fact that the respiratory quotient increases after ingestion of cellulose and hemicellulose as well as of starch and sugar, only the process is much slower than after ingestion of pure carbohydrates. Thus the process of cellulose digestion takes place with the aid of cytase in precisely the same way as the digestion of starch. Cellulose and hemicellulose are transformed into their respective sugars and absorbed as such. The physical and chemical nature of these substances explains why considerably smaller quantities of them are dissolved and absorbed than of the other articles of nutrition.

Absorption of Aqueous and Saline Solutions.—Aqueous and saline solutions are chiefly absorbed in the small intestine, both by the epithelia and by the intercellular connective trabeculæ, mostly in a direction toward the blood stream.

Digestion and Absorption in the Large Intestine.—Generally speaking food is so completely digested and assimilated in the small intestine that only a few remnants of it reach the large intestine.

Any portions of cellulose and hemicellulose which have escaped digestion undergo transformation in the large intestine, notably in the cecum, where with the aid of the intestinal bacteria they are disintegrated through fermentation into carbon dioxid, methane, hydrogen, and fluid fatty acids (butyric and acetic acids). Absorption of aqueous and saline solutions occurs to a large extent in the large intestine as well as in the small.

Intestinal Movements.—The muscular apparatus of the large intestine consists of an exterior layer of longitudinal fibers immediately underneath the serosa, and an interior layer of circular fibers. Between these muscular layers lies Auerbach's nerve plexus. In the submucosa, between the layer of circular fibers and the mucous membrane, is Meissner's nerve plexus. Both these nerve plexuses are autonomous centers. The mucous membrane has besides a very fine muscular layer, the *muscularis mucosæ*, which is supposed to have the special function of protecting against injury from pointed and sharp foreign bodies by means of reflex movements controlled by Meissner's plexus.

The small intestine, when not functioning, is an anemic tube without movement, and in tonic contraction. After the ingestion of food the anse of the small intestine become hyperemic, thicker and shorter, and execute various movements which are differentiated as *segmenting*, *peristaltic*, and *pendulum*.

The *segmenting movements* are continuous advancing contractions of the circular and longitudinal fibers, occurring spontaneously and automatically every five or six seconds as long as there are any intestinal contents. They effect a thorough mixing and kneading of the contents without causing any change in the position of the intestine. Cannon calls this "rhythmic segmentation" and believes that the mixing of the food in the small intestine is dependent upon this movement rather than upon the peristaltic wave. The peristaltic wave forces the food onward. The combined peristaltic wave and segmentation brings every particle of food into contact with the mucous membrane of the intestine, at the same time propelling the contents onward.

The *peristaltic movements* are brought about by a local irritation which causes a strong tonic contraction of the intestinal segment above the place of irritation, while the part below the place of irritation relaxes for a considerable distance. This causes the intestinal contents, from which the local irritation emanated, to be driven downward for some distance. The irritation which produces peristalsis is more intense in proportion as the ingested food is coarse and indigestible. It naturally follows that food very rich in cellulose promotes peristalsis most. However, food remnants difficult of digestion are not the only excitants of peristalsis; the same effect is brought about by the decomposition products which result from the presence of intestinal bacteria (products of

protein decomposition and fermentative acids, formic acid, acetic acid, propionic acid, butyric acid, succinic acid, lactic acid, etc.). Here again the decomposition products of cellulose in the large intestine play the most important rôle. Persons with very good cellulose digestion frequently suffer from chronic constipation. Among the intestinal gases, there are especially carbon dioxide, marsh gas, and sulphuretted hydrogen, which cause marked peristaltic movements. Furthermore, the influence of psychic excitement upon the intestinal movements should not be underrated.

Peculum movements are caused by exaggerated peristalsis in those intestinal segments which are highly charged with fluid contents and gas. In this form of movement the contents of these segments are propelled downward with great velocity and with loud burborygmus for rather a long distance, until the impulse suddenly ceases, only to reassert itself after a more or less prolonged interval. The sudden peristaltic wave, carrying the contents along the whole small intestine, has been called by Meltzer and Auer the "peristaltic rush."

Generally, there is a decrease in the intensity of the movements in the small intestine from above downward; those in the large intestine are usually the same as in the small intestine, but slower. The segmenting movement in the large intestine does not occur so frequently—only at intervals of ten to fourteen seconds—but of greater intensity. The chyle usually passes through the small intestine in two to six hours, while twenty to twenty-four hours, and in many persons a much longer time, is required for it to traverse the shorter large intestine.

Cannon infers from his observations on cats that after the intestinal contents pass from the ileum into the cecum there is a to-and-fro movement from the beginning of the transverse colon to the cecum. This antiperistalsis (*anastalsis*) continues for some time. The tonic ring in the movements of the intestine is of primary importance in the study of anastalsis. In a state of tonus and a locally increased tonic contraction, antiperistalsis of the colon is easily explained. Antiperistaltic movements occur in the cecum, the ascending colon, the proximal part of the transverse colon, and the sigmoid flexure. This antiperistalsis explains why the intestinal contents are retained in the cecum, the ascending colon, and also in the sigmoid flexure, for a much longer period than in the distal segment of the transverse colon and the proximal segment of the descending colon.

The intestinal movements are controlled by Auerbach's plexus through exciting impulses of the vagus and inhibiting impulses of the sympathetic. Great excitation of the sympathetic and its terminations may arrest all the intestinal movements proceeding from Auerbach's plexus and the vagus.

Keith discovered a nodal tissue intermediate between nerve and

muscle and interposed between Auerbach's myenteric plexus and the smooth muscle of the intestinal wall. This intermediate tissue, consisting of branched cells in direct connection with both nervous and muscular elements, bears the same relation to the intestinal musculature as the primitive nodes and conducting tissues of the heart bear to the auricular and ventricular muscular masses. It possesses two distinct functions: one, the initiation and regulation of the muscular contractions in the segment of the intestine which it controls; the other, the power of conducting impulses which lead to the forward propulsion of the intestinal contents. Keith found that food passing along the alimentary tract is propelled through a series of zones or segments furnished with their own pacemaker. To obtain an orderly propulsion of the food along the whole length of the alimentary canal, these various rhythmic zones must be closely coordinated in their action; and this coordination means a complicated system of reflexes (see pages 561 and 696).

It is now believed that the entire digestive tract is supplied with extrinsic nerves which when stimulated cause increased tone and the destruction of which results in loss of tone. Cannon is convinced that this tonus is fundamental. It supplies the resiliency that causes the state of tension when the canal is filled or establishes the state of tension when the canal is only partly filled. This state of tension is indispensable for the contraction of viscera which are walled with smooth muscle holding a nerve net. Hunger contractions have no connection with the normal tonus of the stomach; they are initiated and sustained by the impulses of independent neurons in the central nervous system, and when these neurons become fatigued through prolonged action the hunger contractions of the stomach cease. These contractions are normally continuous in infants; while in adults, even though food be denied, they alternate with quiescent periods.

As a rule the chyme, after having left the stomach, passes through the small intestine in two or three hours; under normal conditions the transverse colon may contain the first food remnants in four hours, and the sigmoid flexure may be filled in six hours. On the other hand, the fecal mass is often retained for hours in the splenic flexure. The movement of the fecal mass has been thoroughly studied by means of the Roentgen ray (see Chapter V). In the cecum and ascending colon the intestinal contents are still soft and massy, while farther on they are of a more formed consistence. The sigmoid flexure is the real fecal reservoir. The presence of fecal matter in the rectum causes a sensation of tenesmus, and the sensitiveness of the rectum is so pronounced that the presence of gases and of solid or fluid feces can be very accurately distinguished.

The closure of the rectum is effected by the sphincter ani internus (smooth muscle fibers) and externus (transversely striated fibers). Both sphincters are continually in a state of tonic con-

traction, which may be increased or inhibited. They possess their own automatic nerve center, but are subordinated to other centers in the spinal column and cerebrum. If a defecation is desired, the cerebrum effects an inhibition of the sphincteric contraction, upon which the fecal column is propelled downward.

Law of Contrary Innervation.— This law, as laid down by Meltzer, is manifest in all functions of the animal body. Stimulation or contraction in any part of the digestive canal induces inhibition or relaxation of the part just below. Contraction of the lower parts of the esophagus and of the cardia is inhibited as soon as the upper end of the canal of deglutition begins to contract. Local stimulation of a segment of the intestine causes contraction at the point of stimulation and relaxation of the part below. This phenomenon has been called by some authors the law of the intestine, but it is now known to be a general law of contrary innervation.

Feces. The feces which are finally evacuated per rectum as an end-product of digestion consist partly of food remnants, partly of remnants of the secretions of digestion, with additions of mucus, bacteria, intestinal epithelia, and excretory products of the large intestine. As is well known, there are also hunger feces in which nitrogen and ash constituents, especially lime, phosphates, magnesium, and even iron, are excreted into the lumen of the intestine. The time for the evacuation of the feces after a given meal varies from eight to thirty hours. It is important to respond as soon as there is a desire for defecation. If the endeavor is not made, the rectum soon becomes accustomed to the presence of feces and fails to sensation for desire.

For further observations on the subject of feces, Chapter IV on Examination of the Feces should be consulted.

CHAPTER II.

EXAMINATION OF THE STOMACH CONTENTS.

EXAMINATION of material obtained from the fasting stomach is one of the most important diagnostic aids in ascertaining the nature and extent of pathologic conditions of the stomach.

The presence of food remnants in large quantities from the last or from a preceding meal, especially if sour-smelling, points to a disturbance of gastric motility. If the quantity of gastric juice which may be removed from the fasting stomach constantly exceeds 100 Cc., a condition known as *gastrosuccorrhœa* (Reichmann's disease), *gastrorrhœa*, hypersecretion, or *gastrochylorrhœa*, is present. According to recent investigations, it is highly probable that *gastrochylorrhœa* is a sequel to disturbance of the motor functions of the stomach. A small amount of mucus and saliva may be found in the normal fasting stomach, its viscosity being observed in pouring from one vessel to another. Numerous mucin bodies and epithelial cells are seen upon microscopic examination. The presence of mucus and saliva in the fasting stomach may be due to stomatitis, pharyngitis, ptyalism, or pathologic conditions affecting the glandular portion of the stomach.

Bile may regurgitate into the stomach from the duodenum. When it has been long in the stomach it undergoes change, its bilirubin becoming biliverdin, so that the fluid takes on a yellowish or greenish color. Bile does not interfere with the peptic activity of the gastric glands, except that, like every protein body, it has a strong affinity for the acid of the stomach. Sometimes in the fasting stomach a mixture of bile, pancreatic juice, and perhaps *succus entericus* is found—without special pathologic significance if the quantity is small.

Blood is found in the stomach under such conditions as hemorrhage from gastric ulcer, irritation of the pathologic mucosa upon the passing of a stomach tube, or vigorous movements caused by expression of stomach contents. Hemorrhages may originate in the mouth, esophagus, pharynx, nasal cavity, or lungs. Hemoptysis and hematemesis may exist simultaneously; when, however, only one is present, it is not a difficult matter to distinguish its source. Slight hemorrhages, with admixture of mucus, are significant only when found upon repeated examinations.

Pus, according to recent investigators, is frequently found in stomach contents; it is always pathologic. It is often found in

cases of ulcerating carcinoma. It is easily recognized, even macroscopically, in such cases, by the foul-smelling yellowish-green and occasionally blood-stained masses.

TEST MEALS.

The secretion of gastric juice starts almost as soon as the food enters the stomach and continues until it enters the duodenum. The investigations of Pawlow show that it starts, as a result of anticipation, even before the food reaches the stomach (psychic secretion). During the latter period of gastric digestion the secretion normally decreases, for which reason the results of analytic examination of the gastric contents are subject to variation. The test hydrochloric acid secreted by the stomach unites with all the protein and salts to form combined acids. Only after all these amounts have been satisfied can we find free hydrochloric acid. If a meal consists of large quantities of protein, it is obvious that free hydrochloric acid will appear later than if the meal consisted in larger proportion of carbohydrates. A test meal should contain all the ingredients of an ordinary meal. In order to make a study of the secretory function of the stomach we must have a standard test meal of definite and constant composition. It is customary to give test meals in the morning, when the stomach is most likely to be empty; occasionally, however, they are given at noon or in the evening, according to the purpose in view.

Ewald-Boas' Test Breakfast.—Ewald-Boas' test breakfast consists of a roll or two slices of white bread without butter and two small cups (300 to 400 C.c.) of water or weak tea without cream or sugar. The patient should thoroughly masticate the bread or roll. The stomach contents should be removed in one hour, since digestion is at its height at this time. This test breakfast contains protein, sugar, starches, non-nitrogenous extractives, and salts. It will thus be seen that the stomach is offered all the usual ingredients of a meal, with the advantage that the whole is liquefied in a very short time and so modified that passage of the contents through the stomach tube is not hindered, as might be the case if more solid food were taken. This test breakfast, while suitable for routine examination, has the disadvantage of introducing into the stomach a variable amount of lactic acid as well as numerous yeast cells with the bread.

Boas' Test Breakfast.—Boas' test breakfast consists of a tablespoonful of rolled oats in a quart of water, reduced to one pint by boiling. A pinch of salt is added to make it more palatable to the patient. This meal, inasmuch as it does not contain lactic acid, is usually given when detection of lactic acid is important, as in cases of suspected carcinoma.

Riegel Test Dinner. At noon the patient is given a meal consisting of beef broth, 150 to 180 Cc. (5 to 6 ounces) of beefsteak, 60 Gm. (2 ounces) of mashed potatoes, and a roll of white bread. The stomach contents are removed in from three to four hours and examined. The advantage of this test meal is the opportunity it affords to note the degree of digestibility of starches and proteins. Fleiner's test meal is similar.

MACROSCOPIC EXAMINATION OF STOMACH CONTENTS.

Having withdrawn the test meal at the allotted time, the physician should carefully inspect the appearance and note the quantity and odor of the material. After the stomach tube is introduced, as stated below, there are two methods of obtaining the stomach contents: (1) The expression method, and (2) aspiration by means of some suction apparatus.

Methods for Obtaining Stomach Contents.—Expression Method.—The first method is the simplest and easiest at our command, and the stomach tube itself is the only instrument necessary. The tube being in the stomach, the patient is instructed to take a deep inspiration, to hold his breath, and bear down with his abdominal muscles, when the gastric contents will pour out from the end of the tube into a tumbler held for their reception. Sometimes coughing or moving the tube a little will produce a gagging sensation, and this induces the abdominal pressure that forces out the stomach contents. Should nothing come through the tube, it may be assumed that the stomach is empty. In removing the tube it is well to cover the end snugly with the finger, to prevent the escape of as much of the stomach contents as the tube contains, thereby adding so much more to the quantity for examination and at the same time avoiding a "muss."

Aspiration Method. For removing gastric contents by the second method, almost any instrument that will create a vacuum may be employed. The so-called "stomach pump" has been used, but it has been found that sometimes, even in its careful use, pieces of gastric mucous membrane are detached—drawn into the eye of the tube. The aspirator bulb of Ewald consists of a ten-ounce Politzer bag, with a large-sized hard-rubber tip over which the stomach tube can be adjusted. The stomach tube having been placed in position in the stomach, the air is forced out of the bag or bulb, and the tube is attached; then, allowing the bag to expand, the stomach contents are aspirated. Aspirating bottles with stopcocks and other complicated attachments have been devised for the removing of the stomach contents, but such apparatus is really unnecessary.

I have an improved stomach tube which I find very practical (Fig. 1). It is made of soft rubber, but, though readily flexible, is nevertheless rigid enough, owing to its size and the thickness of its

walls, to avoid all danger of coiling or kinking in the pharynx or esophagus. It is 84 cm. (32 inches) long, rounded and solid at its lower end, with two lateral openings, one about 2 cm. (three-fourths inch) from the end, the other just above it on the opposite side. These openings run obliquely upward, and have no sharp edges; the edges have been smoothed away in molding (velvet-catheter eye),

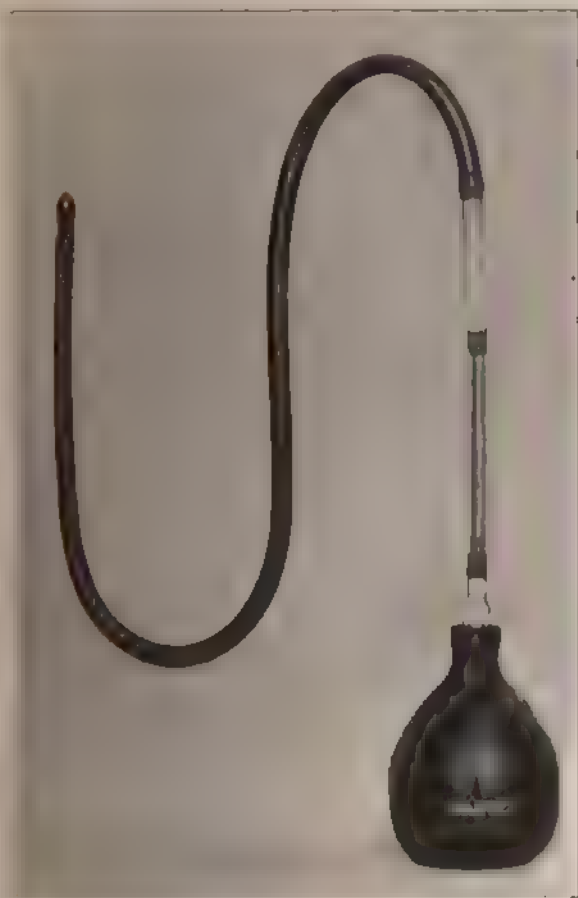


FIG. 1.—Author's improved stomach tube and bulb.

so that traumatism or other injury to the mucous membrane from contact is impossible. The openings are large, rendering aspiration of the stomach contents easy. The lower end is made solid so that no particles of food can accumulate there, as frequently occurs in tubes with a terminal cul-de-sac. The main distinguishing feature of this stomach tube is that the closed, smooth, rounded end contains a heavy piece of lead. This weighted end facilitates the pas-

sage of the tube down the esophagus without any effort on the part of the patient (Fig. 2). Most patients regard the swallowing of a formidable-looking stomach tube as somewhat of an ordeal, but there is no need of swallowing or "gulping" when this tube is used.

In the use of this tube, when the stomach contents are to be aspirated, the patient should sit upright, with his head bent slightly forward and held absolutely motionless. He is instructed to breathe slowly and regularly through the nose and to pay absolutely no attention to what is about to take place. The physician, standing in front of the patient, dips the free end of the tube in cold water and passes it directly to the posterior pharyngeal wall, which guides it downward toward the entrance of the esophagus. The weighted end carries the tube to the laryngopharyngeal opening, where it stops. A gentle push on the tube is now all that is necessary to carry it over the cricoid cartilage down the esophagus into the stomach, provided



FIG. 2. a, Lower end of author's stomach tube, b, sectional view; c, lead, d, solid rubber.



FIG. 3.—Eschhorn's stomach bucket.

there is no obstruction in the esophagus. It is unnecessary to tell the patient to swallow, except in rare instances when the muscles of the neck contract spasmodically, holding the tube tight. Many times the tube passes so quickly into the stomach that it is advisable to move it backward and forward to irritate the throat and induce gagging, which greatly assists the aspiration process.

Before passing the tube, I have the external end connected with an evacuating bulb by means of a short piece of glass tubing and a soft-rubber tube about 16 cm. (6 inches) in length. The bulb is

then compressed, and a bend in the soft-rubber tube held with the thumb and finger of the left hand prevents it from filling with air. As soon as the tube reaches the stomach the soft-rubber connecting tube is released, and the bulb, in recovering its natural expanded form, aspirates sufficient gastric contents for analytic purposes (Fig. 1).

Should lavage be desired, it can be readily and safely carried out with the tube and the evacuating bulb. The food residue in the stomach is aspirated as completely as possible with the empty bulb; the bulb is then detached, filled with water, reattached, and slowly compressed until all the water has been introduced into the stomach. Then, by the mere act of releasing the pressure on the bulb, the water is withdrawn by aspiration from the stomach. This procedure is repeated with a second bulbful, and as often as may be necessary until the water returns perfectly clear.

In dealing with the difficulties the physician encounters in removing the stomach contents of hypersensitive patients, I find that nausea and retching is one of the greatest obstacles to the successful performance of the task. These patients become nauseated at the mere sight or thought of the stomach tube. The very act of cocaineizing the posterior or faucial wall, which some authors advocate to overcome this unpleasant feature of the proceedings, conduces to the production of retching and is by no means willingly tolerated by this class of patients. Some authors, indeed, go so far as to advise abandoning any attempt at introducing the tube in patients with pronounced retching tendencies, dispensing with this method of examination altogether rather than subject such patients to the ordeal. It is evident that the apprehensions of sensitive patients cannot be entirely allayed by persuasion, tact, or skill. The question then arises: Can the unavoidable unpleasantness and inconvenience be shortened, and how? The answer is that it can be shortened and minimized by introducing the tube less deeply—in fact by not allowing it to enter the stomach at all, and yet obtaining a quantity of gastric contents sufficient for chemical and microscopical analysis.

Regurgitation. In devising this process,¹ I have been guided by a consideration of the anatomic fact that the lower third of the esophagus is normally distended, forming a continuous open lumen. As soon as the stomach tube reaches that part, the cardia, following the law of contrary innervation, becomes relaxed, and as a result the gagging and retching of the patient induces a regurgitation of some of the gastric contents into the esophagus, where the fenestrated end of the stomach tube is ready to receive it, and whence it is promptly aspirated by the atmospheric vacuum action of the terminal rubber bulb (Fig. 1). In the mere act of passing beyond

¹ Azon (Charles D.): A Simplified Method of Aspirating Gastric Contents in Hypersensitive Patients, Michigan State Medical Journal, September, 1918.

the tracheal bifurcation the tube causes the contents of the stomach, however low, to regurgitate into the esophagus through the relaxed cardia and meet the receptive stomach tube half-way. Thus it is quite unnecessary for the tube to enter the stomach, and the greater the gagging and retching of the patient the easier it is to obtain a sample of the gastric contents.

When the tube is not in use it should never be coiled, but laid out straight. If this precaution is observed, the original shape of the tube will be maintained so that it will glide easily through the esophagus into the stomach as described.

Einhorn has devised a stomach bucket to remove the stomach contents. It consists of a small capsule-shaped vessel (Fig. 3) made of silver ($1\frac{1}{2}$ cm. long, $\frac{3}{4}$ cm. wide), open at the top and for a short distance down the side. The opening is surmounted by an arch, to which a silk thread is tied, and a knot is made 16 inches from the attachment. In order to secure a sample of the stomach contents, the bucket is first dipped in lukewarm water (filled and emptied) to facilitate filling when in the stomach; the patient is asked to open his mouth wide, and the bucket is placed on the root of the tongue (almost in the pharynx); the patient is then instructed to perform the act of swallowing, and within a few seconds the bucket enters the stomach. It is left there for five minutes and then withdrawn. During the withdrawal of the apparatus, resistance is usually felt at the introitus esophagi. To overcome this difficulty the patient is again instructed to swallow, by which act the larynx is pushed forward and upward in such a manner as to free the passage, when the bucket can be easily withdrawn. If the stomach was not empty, the bucket returns with gastric contents sufficient for the making of various important tests.

Inspection of Stomach Contents.—By inspection one should distinguish between absolutely undigested, partially digested, and well digested contents. It is also possible to distinguish by inspection between carbohydrate and protein digestion. Absolutely undigested food masses are found in advanced cases of gastric catarrh, in atrophic conditions of the gastric mucous membrane, and likewise in achylia gastrica. The presence of undigested food points also to marked secretory disturbance. In such conditions the appearance of the test meal after removal resembles that of a mixture of bread and water before ingestion. The absence of peptic digestion is ascertained by the clearness of the filtrate. By inspection the presence of blood, mucus, bile, or intestinal juices, and occasionally pus, animal parasites, and fragments from the gastric mucosa, may be detected. In cases characterized by marked gastric retention, the stomach contents when placed in a vessel and allowed to stand for a few minutes are sometimes observed to be in three separate layers: the upper consists of mucus, or undigested food particles which have undergone fermentation; the next, which

is the largest, of fluid; while that on the bottom of the vessel consists of chyme. This is the condition found in abnormal gastric fermentation and extreme motor insufficiency (dilatation).

The filtrate of the entire contents of the normal stomach, evacuated exactly one hour after a test breakfast, measures 20 to 50 Cc. There may be much less than this, or the stomach may be entirely empty; if so, the condition is what has been designated hypermotility or hyperkinesis, found in organic and nervous gastric affections, such as chronic gastritis, achylia gastrica, bulimia, and whenever there is insufficiency of the pylorus. On the other hand, if remnants of the preceding meal are constantly found in the stomach contents in the morning, the finding is indicative of impairment in gastric motility, the degree of which can be ascertained only by repeated examinations of the stomach contents.

Determination of Gastric Juice. The method of Mathieu and Rémond is commonly used to determine the total amount of gastric juice secreted. The gastric contents are removed as completely as possible at the usual interval after an Ewald test breakfast. Water, 200 Cc. ($\frac{1}{2}$ pint), is then poured into the stomach through the stomach tube and thoroughly mixed with the remnants of the test breakfast in the stomach by moving the funnel up and down, as well as by pressure upon the stomach. As much as possible of this mixture is then evacuated in a separate receptacle, and the clinician proceeds to ascertain the acidity of the undiluted as well as that of the diluted stomach contents. From these data, conclusions may be drawn as to the degree of dilution and the amount of the residual gastric contents.

Mathieu endeavors to ascertain the total stomach contents by the following formula:

- a = the acidity of the undiluted gastric contents.
- b = the acidity of the diluted gastric contents.
- x = the amount of the test meal remaining in the stomach after the first extraction

200 Cc. = the amount of water introduced into the stomach for diluting.

Then

$$\begin{aligned} a : b &:: (x + 200) : x \\ ax &= b(x + 200) \\ ax - bx &= 200b \\ x &= \frac{200b}{a - b} \end{aligned}$$

In ascertaining the acidity of the stomach contents, it is necessary to determine the total available acidity rather than the mere degree of acidity.

COLOR.—Gastric juice is a colorless liquid, though at times faintly opaque. It may vary, however, with the color of food taken. Coffee or particles of toasted bread will lend a distinctly brownish coloration, while meat will tend to discolor the juice red. A distinct red color may also be due to the presence of blood, which grows darker the longer the blood remains in the stomach. The color of

the gastric contents may be either yellow or green, due to the presence of bilirubin or biliverdin, biliary pigments which may be detected by the tests for bile in the urine. A brownish-black coloration and fetid odor of the stomach contents points to intestinal obstruction below the duodenum.

Odor.—The odor of normal gastric juice is slightly sour. It is offensive when the gastric juice is mixed with materials from the intestinal canal. In the vomitus of uremia there is often a distinct odor of ammonia; an alcoholic odor is present in alcoholic intoxication. Stagnation of gastric contents gives rise to an intensely strong odor.

Consistency.—Usually watery in character, the normal stomach contents vary with the character of the extraneous material composing them. In catarrhal gastritis or in cases marked by subacidity, there may be present after a test meal so much tough, slimy mucoid material as to render filtering of the stomach contents impossible.

The stomach is practically never empty, always containing a certain quantity of fluid, normally acid in reaction, in amounts of not less than ten or more than a hundred cubic centimeters.

CHEMICAL EXAMINATION OF STOMACH CONTENTS.

Chemical examination of gastric contents consists in the use of reagents to determine the actual state of digestion, so that by comparing it with normal physiologic digestion one may obtain information in regard to any functional disturbances or changes present. These examinations should be made as frequently as may be necessary to enable the clinician to form a correct estimate of the condition of the gastric function; it is only in rare cases that positive results can be obtained from a single examination.

Apparatus.—The special apparatus required for the analytic work is very simple (Fig. 4).

In a complete chemical analysis the following tests should be made:

Tests	Reagents
1. Reaction	Litmus.
2. Hydrochloric acid	Ganzburg (see p. 76)
3. Total acidity	Phenolphthalein (see p. 79).
4. Free hydrochloric acid	Dimethylamidoazobenzol (see p. 80).
5. Combined hydrochloric acid	Alzarin (see p. 81)
6. Lactic acid	Uffelmann (see p. 82)
7. Pepsin	Mett (see p. 84).
8. Rennin	Calcium chloride (see p. 84).
9. Propeptone	Copper sulphate (see p. 84).
10. Peptone	Sodium chloride (see p. 84).
11. Dextrin	Lugol solution (see p. 85).
12. Erythro-dextrin	Lugol solution (see p. 85).
13. Achroo-dextrin	Lugol solution (see p. 85).
14. Maltose	Fehling solution.

Determination of Reaction.—After macroscopic examination of the stomach contents, a portion should be filtered and the filtrate tested with litmus paper, in order to ascertain the reaction, which may be acid, alkaline, amphoteric, or neutral. If the reaction is found to be acid, the next step is to ascertain the presence of free hydrochloric acid. This is done by means of Congo red. Congo red was introduced into practice and recommended in the form of Congo paper, as a reagent for free hydrochloric acid. Congo red in solution is, however, more sensitive than Congo paper. The solution is prepared by dissolving 1 gram of the powdered Congo red in 100 Cc. of water. By the use of the solution 0.0009 per cent. of hydrochloric acid may be detected, while the paper does not react unless 0.01 per cent. of acid is present. Congo red paper consists simply of filter paper saturated with an alcoholic solution of Congo red and allowed to dry. The color changes from red



FIG. 4. Necessary apparatus for making analysis of stomach contents: A, glass tumbler for holding stomach contents, B, filter paper, C, glass funnel; D, sedimentation glass, E, gastric filtrate, F, F, graduated pipets, holding 5 Cc., G, porcelain spoon, H, beaker, I, alcohol lamp, J, buret for titrating with one-tenth normal sodium hydrate solution, K, buret stand.

to blue on contact with free hydrochloric acid. The test establishes the presence of free mineral acids only. High acidity from free lactic acid in the stomach may, however, give a distinct reaction. The test is of value for the detection of free hydrochloric acid, since this is ordinarily the only mineral acid to be found in the stomach contents.

Dimethylamidoazobenzol Test. This test depends upon the coloration which a 0.5-per-cent. alcoholic solution of dimethylamidoazobenzol produces when treated with gastric juice containing free hydrochloric acid. To make the test, a few cubic centimeters of filtrate from the stomach contents after the test breakfast are placed in a porcelain spoon or dish, and one to two drops of the dimethylamidoazobenzol solution added. A carmin red color results when free hydrochloric acid is present (Plate II, Fig. 1). This reagent does not react to organic acids unless they are present in amount over

0.5 per cent. The proportion of free hydrochloric acid present may be determined by the intensity of coloration when the reagent is added, for as small a proportion as one part to fifty thousand, or 0.002 per cent., gives the color reaction.

From a clinical point of view it is of the utmost importance to determine the presence or absence of hydrochloric acid. After this has been determined, it must be ascertained whether the secretion is increased or decreased. When free hydrochloric acid is found to be present, it is unnecessary to test for pepsin or pepsinogen, since this ferment is always present when free hydrochloric acid can be demonstrated. When, however, this acid is absent, we may still have a secretion of pepsinogen.

For the detection of free hydrochloric acid the Günzburg test is perhaps the most reliable.

Günzburg's Test.—Günzburg's, or the phloroglucin-vanillin, reagent is prepared as follows:

	Gm. or Cc.	
R Phloroglucini	2 0	3ss
Vanillini	1 0	gr. xv
Alcoholis absoluti	30 0	5j
Misce.		

Three drops of filtered stomach contents are placed in a porcelain spoon or dish (Fig. 4, a); to this 3 drops of the reagent are added from a small pipet, and the two solutions are thoroughly mixed. The porcelain spoon or dish is then very carefully heated over a small flame (Fig. 4, 1), when if free hydrochloric acid is present a cherry red tint is obtained around the edges of the mixture (Plate 1, Figs. 3 and 4). This color is due to the deposition of very fine crystals, an effect which would occur in even aqueous solutions of 0.01 per cent. of the reagent. This peculiar color is not developed by any organic acid whatsoever.

Instead of the phloroglucin solution, a filter paper prepared by means of it is sometimes used; when moistened with two or three drops of stomach contents and heated, it reveals the presence of hydrochloric acid by developing the same cherry-red tint. The test with the solution is more reliable.

QUANTITATIVE ANALYSIS.

The buret is used for all quantitative analyses. It is graduated in tenths of a cubic centimeter, to be easily read. The buret should be fixed in a perpendicular position and firmly attached to its stand (Fig. 4, x). It should be filled through a glass funnel with the solution to be used. Care must be exercised to avoid the presence of air bubbles. The buret is graduated from zero to 30 Cc. Allow enough of the solution to run out to remove the bubbles and



to bring the solution down to the zero mark. In reading off the quantity of solution that has been used, great care should be taken to read at the level of the bottom of the meniscus formed by the attraction of the fluid to the cylindric wall of the buret.

Normal Solutions. For quantitative analysis of the acid in the gastric contents, normal solutions are used. A normal solution of acid or alkali is one in which each liter represents the number of grams of reagent resulting from dividing the molecular weight of the substance by the number of replaceable hydrogen atoms or hydroxyl groups. A decinormal solution is one-tenth the strength of the normal solution. It is this latter that is used in making stomach analyses. In the various tests employed in quantitative analysis for acidity of the gastric contents, one-tenth normal sodium hydrate is used in the buret. The amount of this alkali necessary to neutralize a given quantity of the acid in the gastric juice will give the degree of acidity. It has been found that the normal acidity of the stomach contents at the height of digestion (one hour after a test breakfast) will range between 40 and 60 degrees, which means the number of cubic centimeters of one-tenth normal sodium hydrate solution necessary to neutralize 100 cubic centimeters of gastric juice. For example, if we use 2.5 Cc. of one-tenth normal sodium hydrate solution to neutralize 5 Cc. of gastric juice, the degree of acidity would be $2.5 \times 20 (= 50)$. We multiply by 20 because we always figure on the amount necessary to neutralize 100 Cc. of gastric juice, and since we have used only 5 Cc. for the test we must multiply by 20 to bring this up to 100.

One cubic centimeter of one-tenth normal sodium hydrate solution will neutralize 0.00365 gram of free hydrochloric acid. If now we multiply this factor by the number of cubic centimeters necessary to neutralize 100 Cc. of the filtered gastric juice (degree of acidity), the result will be the percentage of acid present. If the normal acidity is between 40 and 60 degrees, the percentage will be found by multiplying by 0.00365.

Minimum normal acidity 40 degrees, $0.00365 \times 40 = 0.146$ per cent.

Maximum normal acidity 60 degrees, $0.00365 \times 60 = 0.219$ per cent.

After the Ewald-Boas test breakfast an excess of free hydrochloric acid should be present within fifty or sixty minutes, while after a Riegel test dinner it is present in from two and a half to three hours.

The elements to which the acid reaction of stomach contents is attributable are outlined in the following table:

1	Hydrochloric acid	
	free	combined (with proteins, basic substances)
2.	Organic acids (lactic, butyric, acetic acids)	
	free	combined (with proteins, basic substances)
3.	Acid phosphates.	

Since the normal acidity of the stomach contents is between 40 and 60 degrees, clinicians have for the most part agreed that above 60 degrees shall constitute hyperchlorhydria, hyperacidity, or superacidity; below 40 degrees, hypochlorhydria, hypoacidity, or subacidity; absence of acid, achlorhydria, anacidity, or achylia. The *total* acidity is ascertained by the phenolphthalein test (see page 79).

The unfiltered gastric contents reveal, on analysis, a higher acidity than the filtered, and it has been suggested that such an analysis would furnish a better indication of the condition of gastric secretion than analysis of the filtered contents. Inasmuch, however, as the work of Ewald and Boas in establishing hydrochloric acid values was done on filtered contents, a change would necessitate new standards, involving a tremendous amount of work.

Fractional Analysis.—In fractional determination of gastric secretion, specimens of the gastric contents are withdrawn at fifteen-

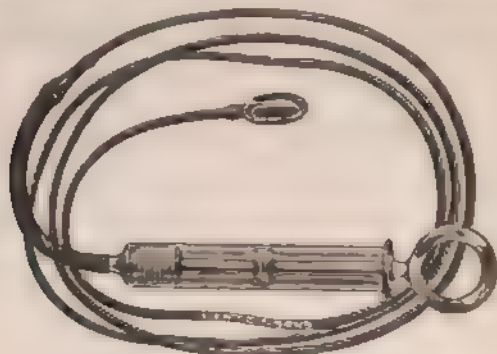
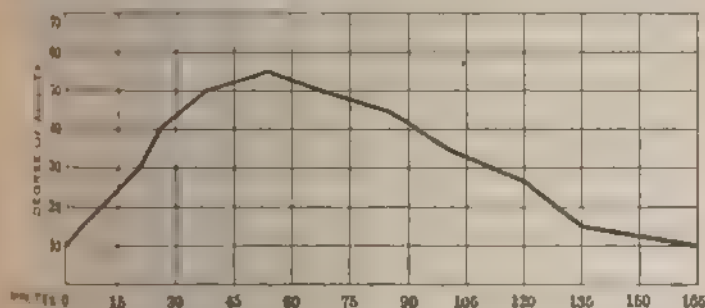


FIG. 5.—Rehfuess gastroduodenal tube.

minute intervals during the cycle of digestion, and the free, combined and total acids are titrated. For withdrawing the stomach contents for examination, either the duodenal tube described on page 98 or a Rehfuess stomach tube may be used. The Rehfuess instrument is an ordinary rubber tube with a metal end having slit-like openings, each equal in width to the inner diameter of the tube (Fig. 5). Aspiration is greatly facilitated by the size of these openings. The patient takes an ordinary Ewald-Boas test breakfast, and ten minutes later the tube is swallowed with two ounces of water. The patient is placed at ease, either sitting or lying down, and urged to read to divert his attention. At intervals of fifteen minutes 15 to 20 Cc. of the stomach contents is withdrawn until the stomach is empty. By testing each portion, the whole cycle of digestion is recorded (Fig. 6).

The motor power of the stomach is demonstrated by the disappearance of all food particles from the fifteen-minute samples.

By this method of examination it is possible to follow the entire cycle of gastric digestion with practically no discomfort to the patient and to draw off at any moment a sufficient quantity of the gastric juice for the necessary chemical examinations. The principle of the entrance and retention of the Rehfuess tube is gravity; the tip is sufficiently heavy to seek the lowest portion of the stomach. The instrument is left in the stomach for hours, or until the gastric cycle is completed. A curve can be constructed which graphically records the entire course of digestion. Information as to the amount of the secretion can be obtained at any point in the progress of digestion, by complete aspiration and noting the character of the specimens obtained. There is no specific curve for the normal person; but three types of curve can be found, illustrating the rapidity of reaction to a given stimulus, the height of the curve, and its descent. These types have been termed, respectively, the hypersecretory, the hyposecretory, and the isosecretory.



13-1—A fractional test breakfast secretory curve, showing the degree of acidity in a normal case.

The value of the interval method of gastric analysis is obvious. While the ordinary method of examining the stomach contents one hour after the test meal is taken gives evidence of only a single moment in an ever-changing cycle of gastric activity, the fractional determination shows (a) stomach secretions whose curves fall toward the end of gastric digestion; (b) stomach secretions whose curves rise at the end of gastric digestion; (c) stomach secretions absent or delayed. By fractional analysis of the stomach secretions a diagnosis of hyperchlorhydria, hypochlorhydria or achylia is more firmly established than by the one-hour stomach examination which gives no evidence of the secretory curve.

Phenolphthalein Test for Total Acidity.—The total acidity is determined with one-tenth normal sodium hydrate solution in the buret. The indicator consists of a 1-per-cent. alcoholic solution of phenolphthalein. Draw into a graduated pipet 10 Cc. of the filtered gastric juice (Fig. 4, F). Pour the contents of the pipet into a

beaker (Fig. 4, *u*). To this, add three or four drops of the phenolphthalein solution, which will cause a grayish clouding (Plate I, Fig. 1). The one-tenth normal sodium hydrate solution is gradually added until red is discerned at the point where the solution from the buret touches the gastric juice. By agitation, the red color disappears. Add more of the sodium hydrate solution and again agitate the contents of the beaker. When the reddish color ceases to disappear, a sufficient quantity of the one-tenth normal sodium hydrate solution has been added to neutralize the total acidity of the stomach contents. Care must be taken not to add too much. The end of the test shows a slight red (Plate I, Fig. 2). It is now necessary to read on the buret the amount used. If we have used 4.5 Cc., we multiply by 10, because we calculate the amount necessary to neutralize 100 Cc., whereas we have used only 10, and we find that our acidity is 45 degrees. The percentage of acidity is ascertained by multiplying the 45 by 0.00365, making 0.16425 per cent.

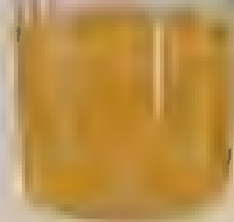
It is very important to make a quantitative estimate of free hydrochloric acid in studying all pathologic conditions of the stomach. When, however, the amount of free hydrochloric acid is diminished, it is necessary to exercise caution in the interpretation of either qualitative or quantitative tests for free hydrochloric acid. In comparatively rare cases all the indicators, with the exception of Günzburg's reagent, have given a positive reaction for hydrochloric acid when no hydrochloric acid was actually present.

Töpfer's Method of Quantitative Analysis.—This is the simplest and most delicate of tests for free hydrochloric acid. One-half per cent. dimethylamidoazobenzol alcoholic solution is used as an indicator. The titration of the filtered gastric juice is done with decinormal sodium hydrate solution. Lactic acid will not react to the test unless it is present to the extent of 1 per cent., which is rarely the case. Acetic and butyric acids are present in fairly large amounts in fermentative processes of the stomach; when present in sufficient quantities to interfere with the reaction for hydrochloric acid, their strong odor renders them easy of detection. To 10 Cc. of the filtered gastric juice, one or two drops of indicator are added; if hydrochloric acid is present, a bright red tone results (Plate II, Fig. 1), so the mere presence or absence of hydrochloric acid is easily determined. The quantitative determination is now made by adding decinormal sodium hydrate solution; as this solution is added, the reddish tint of the mixture changes to a distinct yellow. The titration must proceed to the point at which all trace of red disappears and the color becomes clear yellow (Plate II, Fig. 2). To ascertain the amount of free hydrochloric acid present, note the number of cubic centimeters of decinormal sodium hydrate solution used from the buret. Multiply this by 10, in order to determine the amount necessary to neutralize 100 Cc. of gastric juice—the figures also representing the

PLATE 11

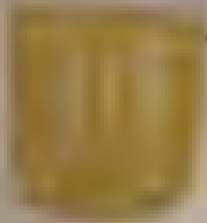


Topley Test



Topley Test

Control Test
Control Test
Control Test



Topley Test



Topley Test

A series of tests showing
the effect of different
conditions on the
Topley Test

Lactic Acid.--Since bread, milk and meat contain lactic acid, any test for lactic acid can be of value only when the meal contains very little of these foods. The Boas test meal (page 67) is preferable when the object is to detect the presence of lactic acid. Under physiologic conditions no appreciable amount of lactic acid is formed during digestion. Lactic acid is apt to be found in any condition

associated with stagnation of the gastric contents as a result of motor insufficiency, provided the amount of hydrochloric acid is below normal. An excess of lactic acid would suggest gastric carcinoma, though it should not be overlooked that an excess of lactic acid may be present in benign stenosis of the pylorus and motor insufficiency. Should the stomach be washed out the evening before, and lactic acid appear in the stomach contents after the night's fast, the pathologic condition is probably carcinoma. Where carcinoma has developed from the overhanging edge of a callous ulcer, the findings may show no lactic acid, but, on the contrary, large amounts of hydrochloric acid.

Uffelmann's Test. Uffelmann's reagent consists of 10 Cc. of a 4-percent. carbolic acid solution to which are added one drop of ferric chlorid solution U. S. P. and sufficient water to produce a transparent amethyst blue (Plate III, Fig. 1). The solution should be freshly prepared for each test. Add a few drops of the filtrate from the stomach contents after a Boas test meal (p. 67) to 5 Cc. of the Uffelmann reagent in a test tube, and in the presence of lactic acid the solution will lose its blue color and take on a beautiful canary yellow or greenish-

yellow tint (Plate III, Fig. 2). Should there be considerable hydrochloric acid present in the gastric juice the result may be obscured. The stomach contents under this condition should be extracted with ether, which takes up the lactic acid only. The ethereal solution is then evaporated, the residue taken up with distilled water, and the Uffelmann test applied to this solution; if lactic acid is present, the solution turns intensely green.

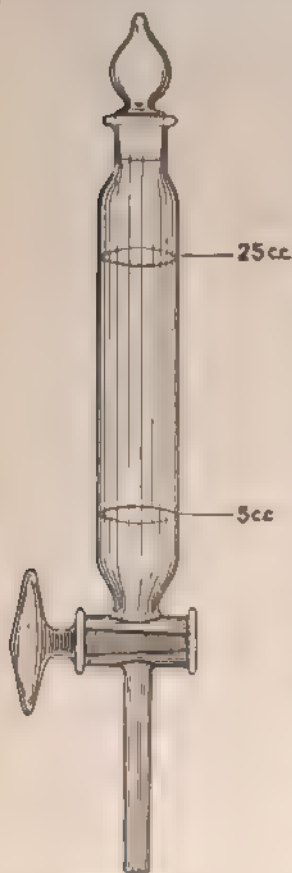


FIG. 7. Strauss' funnel for making lactic acid test.

P. A. H. 111

Strauss' Test. Strauss has devised a glass funnel (Fig. 7) which makes the test for lactic acid quite simple. The funnel is graduated to 5 Cc. below and 25 Cc. above. It is filled to the 5-Cc. mark with filtered gastric juice, and ether is added up to the 25-Cc. mark. The funnel is corked and thoroughly shaken. After standing for a short time to allow the fluids to separate, the contents are allowed to run out through the stopcock to the 5-Cc. mark. Distilled water is added up to the 25-Cc. mark, and then two drops of tincture of iron chlorid. On shaking the mixture, if an appreciable quantity of lactic acid is present an intense green color results; a pale green indicates a trace of lactic acid.

EXAMINATION FOR ENZYMES.

Pepsinogen and Pepsin.—Through the action of acids, and especially hydrochloric acid, pepsinogen is converted into active pepsin. If the gastric contents contain free acids and digested proteins, pepsin is present. If there are no free acids, but the digestive power becomes apparent when the material is treated with sufficient hydrochloric acid, pepsin is demonstrated. To ascertain the presence of pepsin when free hydrochloric acid is present, 10 Cc. of gastric contents is placed in a test tube, a little disk of coagulated egg protein added, and the test tube placed in an incubator, which is kept at a constant temperature between 98° and 100° F., disappearance of the egg protein after a short interval points to the presence of pepsin.

When hydrochloric acid is absent, pepsinogen alone may be found in the stomach contents. This is important to the diagnosis, inasmuch as pepsinogen is rarely absent. The absence of pepsinogen means atrophy or achylia. In the absence of hydrochloric acid, pepsinogen is practically inert. The test for pepsinogen is made by adding to 10 Cc. of filtered gastric juice one or two drops of hydrochloric acid and proceeding as with the qualitative test for pepsin.

Determination of Pepsin.—*Ricin Test (Jacoby-Solms Method).*—One gram of ricin is dissolved in 100 Cc. of a 5-per-cent. solution of sodium chlorid, and the whole filtered. Two cubic centimeters of the filtrate are mixed with 0.5 Cc. of a decinormal HCl solution, 1 Cc. of diluted stomach contents is added, and the mixture is maintained at body temperature for three hours. Ferments clear up the ricin deposit. The quantity of pepsin is determined from the degree of dilution in which the stomach contents will cause the ricin deposit to disappear. Solms considers *one pepsin unit* the amount of gastric juice which is sufficient to clear up 2 Cc. of a 2-per-cent. ricin solution in three hours at blood temperature. Normal stomach contents contain about 100 pepsin units to the cubic centimeter.

Mett's Test. A capillary glass tube is used, into which fresh egg protein is drawn by suction. The contents of the tube are coagulated by immersion for five minutes in boiling water. By cutting the tube into pieces 2 to 5 centimeters long the pieces can easily be placed in a beaker containing the gastric juice to be tested. They should then be kept in an incubator for ten hours at a temperature of 95° to 98° F. At the end of this time the protein will be seen to have disappeared from the ends of each piece, while there still remains some in the middle portion of each. The empty ends are measured. The square of the length of the column of protein digested is the measure of the amount of pepsin in the gastric juice. For instance, if the empty portion of the tube be 3 millimeters in length the digestion equals 3×3 , or 9 parts of pepsin. The peptic unit is that quantity of pepsin which will digest one millimeter of egg protein in a Mett tube in ten hours, the tubes being immersed in 0.18-per-cent. free hydrochloric acid.

The Gelatin Test.—Ramond, Petit and Carrié describe a simple procedure showing both the peptic power and the hydrochloric acidity of given samples of gastric juice. Five Cc. of 3-per-cent. gelatin solution is placed in a test tube 15 millimeters in diameter; the tube is then sterilized at 100° C., and kept exactly vertical during solidification of the gelatin. Five Cc. of filtered gastric juice is then placed over the gelatin, and 0.02 Gm. of thymol added. Allowed to stand at room temperature, a few hours later the tube shows a definite ring in the gelatin, the time of its appearance depending upon the acidity of the specimen of gastric juice. In an acidity or marked hypoacidity no ring forms. To ascertain the rapidity of digestion, a paper index may be pasted along the tube or the latter may be graduated in millimeters. Normal gastric juice digests, on an average, two and one-half millimeters of gelatin in twenty-four hours. The digestion continues regularly until all the gelatin has been consumed. Several tubes should be used for each specimen, and an average taken. The acid ring in the gelatin is most clearly seen when the tube is held against the light.

Qualitative Test for Rennin.—Five to ten cubic centimeters of filtered stomach contents are accurately neutralized with decinormal sodium hydrate solution. The same quantity of neutral or amphoteric boiled milk is added and the mixture placed in an incubator. If the curdling process begins within fifteen minutes and a coagulum is formed when the mixture is allowed to stand longer, the phenomenon of coagulation is attributable to the action of rennin.

To test for rennin zymogen, add three to five drops of a 1-per-cent. calcium chlorid solution to 10 Cc. of milk to which three to four drops of gastric filtrate have been added, and place in an incubator. If coagulation of casein occurs in the course of a few minutes, rennin zymogen is present.

A pronounced diminution of the specific biologic action of fer-

ments is directly indicative of disturbance of the function of the glandular apparatus of the stomach itself. By examination of the gastric enzymes it may be determined in individual cases whether impairment of the glandular apparatus is transitory or permanent. Hydrochloric acid secretion is sometimes temporarily inhibited in anomalies of menstruation, in appendicitis, in nervous dyspepsia, in cholelithiasis, and in acute and the early stages of chronic gastritis. In these conditions the presence or absence of enzymes indicates whether the physician has to deal with only a temporary suppression of the hydrochloric acid secretion or with an advanced or chronic gastric catarrh.

Test for Propeptone.—The end-products of protein digestion in the stomach are propeptones and peptones. The aminoacids are all formed in the intestine. To test for propeptone, mix equal parts of the filtered stomach contents and a saturated solution of sodium chlorid. A turbid precipitation indicates the presence of propeptones. When there is no precipitation, but the addition of two or three drops of acetic acid turns the liquid turbid, propeptone is present. When the solution is heated the turbidity clears up, and when it cools the turbidity returns. The more turbid the solution, other things being equal, the greater the amount of propeptone present.

Test for Peptone.—After having filtered out the propeptone, 5 Cc. of the filtrate is made strongly alkaline by adding sodium hydrate solution. A few drops of a 1-per-cent. sulphate of copper solution are added. When peptone is present, a purple or violet-red color (biuret reaction) appears.

CARBOHYDRATE DIGESTION IN THE STOMACH.

The conversion of starches into sugar occupies three intermediary stages, which are determined by their behavior toward Lugol's solution. The stages are amidulin, erythroextrin, and achroöextrin (see page 50). With Lugol's solution, amidulin gives a blue color, erythroextrin a violet or mahogany brown, achroöextrin remains unchanged. The end-product of the conversion of starch into sugar is maltose, together with small amounts of dextrose, which may be demonstrated by Fehling's or Nylander's tests. Lugol's solution consists of pure iodine, 1 gram; potassium iodid, 2 grams; distilled water, enough to make 20 Cc.

In hyperchlorhydria the digestion of starch has been found to be considerably impaired; in testing the stomach contents for starch, iodine gives a pronounced blue coloring. In achlorhydria the reaction is wine-yellow. The first result is more likely to be obtained when there is an impairment of the salivary glands whereby the secretion becomes poor in ptyalin. In any case where hyperacidity is present, salivary digestion stops as soon as the food enters the

stomach; in subacid conditions salivary digestion may proceed indefinitely in the stomach, depending, of course, upon the extent of the diminution of gastric secretion.

BLOOD IN THE STOMACH CONTENTS.

The presence of blood in the stomach contents must be considered always pathologic; it is most frequently associated with erosion, gastric ulcer, or gastric carcinoma. It may be due to the rupture of varices in the lower part of the esophagus caused by cirrhosis of the liver. In gastric ulcer the blood is usually bright red in appearance, unless changed by the action of the acid of the gastric juice, in which case it takes on a brownish discoloration. In hemorrhages resulting from gastric carcinoma the blood is more thoroughly incorporated with the stomach contents, giving rise to the so-called coffee-ground material, of brownish-black appearance.

Weber's Guaiac Test. A small quantity of the gastric filtrate is rubbed up with water; one-third its volume of glacial acetic acid is then added and the mixture shaken up with ether in a test tube. The acetic acid decomposes hemoglobin and liberates hematin, which is in turn taken up by the ether. The clear supernatant ether is then poured off, ten drops of an alcoholic solution of resin of guaiac are mixed with it, and lastly, twenty to thirty drops of turpentine or Huehnfeld's reagent are added. A blue color appearing at once points to the presence of blood in considerable quantity. Delayed appearance of the blue color is an indication of smaller quantities of blood.

TESTS FOR CARCINOMA.

Salomon's Test.—The principle underlying this test is the fact that carcinoma secretes protein, which becomes mixed with the gastric contents. The diet of the patient for twenty-four hours prior to the test should be absolutely free from protein. At the beginning of this period he is given a morning meal of milk and gruel and a mid-day meal of bouillon with coffee or tea. Late in the evening the stomach should be washed out with large quantities of pure water until the return water is clear. The following morning the fasting stomach is washed twice with 400 Cc. of physiologic salt solution, the same solution being used each time. This solution is then tested by the Kjeldahl method for the total amount of nitrogen, and by Eshbach's method for the quantitative estimation of protein. Salomon found in cases of gastric carcinoma 20 to 70 milligrams of nitrogen and from 0.00625 to 0.05 per cent. of protein to each 100 Cc. of the fluid that had been used in lavage. In non-malignant cases, according to this investigator, no protein could be detected, and the amount of nitrogen varied from none to 16 milligrams in each 100 Cc.

Wolff and Junghans Test.—This test depends upon estimation of the amount of soluble protein present in the gastric contents, and is one of the most reliable in use. The assumption is that a gastric carcinoma secretes a peptid-splitting ferment that converts insoluble protein into soluble protein. The test is useless when hydrochloric acid is present in the stomach, and is therefore of value only in cases of achlorhydria. Wolff and Junghans found large quantities of soluble protein in the gastric contents in cases of carcinoma, while in benign cases of achlorhydria only minute quantities are found. The soluble protein is demonstrated by precipitation with the following reagent:

	Gm. or Cc.	
R - Acidi phosphotungstici	0 3	m v
Acidi hydrochlorici	1 0	m xv
Alcoholis	20 0	3 v
Aquæ destillatæ q. s. ad	200 0	3 vij
Misce.		

This reagent should be kept in a cool place.

Six test tubes, holding varying quantities of filtered gastric juice, from 0.0025 Cc. to 1 Cc., are diluted up to 10 Cc. with distilled water. With a pipet 1 Cc. of the reagent is carefully placed upon the contents of each test tube. At the junction of the reagent and the diluted gastric juice a definite white ring must appear in all six tubes for the test to be positive, for in normal cases it will appear in dilutions containing as much as 1 part of gastric juice to 40 of water.

Cytodiagnosis.—Loeper and Binet devised the cytodagnosis of carcinoma of the stomach. The method consists in washing out the stomach by introducing $\frac{1}{2}$ liter ($\frac{1}{2}$ pint) of normal saline solution and removing it, centrifugalizing, and examining for carcinomatous cells. The solution should reach all portions of the gastric mucosa. With the solution in the stomach, the patient should lie down and sit up several times and the organ be carefully kneaded. Enough freshly loosened carcinomatous cells can thus be obtained to yield a positive diagnosis.

Glycyltryptophan Test.—Neubauer and Fischer state that carcinoma of the stomach secretes a ferment which, unlike pepsin, splits glycyltryptophan, and that this ferment can be detected in the stomach contents in the early stages of gastric carcinoma. On the other hand, it has been shown by Warfield that saliva, when mixed with neutral or faintly acid gastric juice, imparts to the latter the power of separating tryptophan from glycyltryptophan. Jacque and Woodlyatt affirm as a result of their later investigations that saliva free from bacteria does not split glycyltryptophan; also that normal gastric juice free from blood, bile, and bacteria, and gastric juice from cases of benign subacidity, have no peptid-splitting action. In the glycyltryptophan test the presence of tryptophan is manifested by the development of a rose-red color in the gastric contents

under test on the gradual addition of bromin vapor. A weak solution of calcium hypochlorite may be used instead of bromin vapor, and gives the same reaction.

Glusinski's Test.—This test is based upon the assumption that gastric carcinoma is always accompanied by a chronic gastritis which interferes with the secretion of the gastric juice. Examination of the stomach contents after three different test meals in the course of one day reveals whether the gastric juice is being normally secreted. After the first test meal the percentage of hydrochloric acid may be normal, but if the case is one of gastric carcinoma a smaller amount of free hydrochloric acid will be found after the second test meal, and after the third the percentage of hydrochloric acid will be found to be greatly diminished or nil. The test is remarkably reliable in differentiating between carcinoma and ulcer, since the latter does not interfere with the secretion of hydrochloric acid.

INDIRECT METHODS OF GASTRIC ANALYSIS.

A number of methods are in vogue for the examination of the functioning powers of the stomach without removing the gastric contents. While such methods fail to determine the exact condition of the acidity or of the activity of ferments, much may be learned by means of them regarding gastric motility as well as the digestive powers of the stomach.

Benedict's Effervescence Test for Acidity.—When the stomach tube cannot be introduced, the effervescence test for gastric acidity, first described by A. L. Benedict, is of great value. The test is only approximate, and it is not necessary to insist upon a rigid test meal. It consists in auscultation of the effervescence produced when a saturated solution of sodium bicarbonate meets the gastric contents. One hour after a test breakfast or three hours after an ordinary meal, the patient standing before the physician and cautioned not to speak, breathe heavily, or rustle his garments, drinks 30 Cc. (1 ounce) of the sodium solution at one gulp. In from ten to thirty seconds a fine crepitation is heard, the stethoscope being placed over the center of the gastric area. If the gastric acidity is high, the crepitation is quite marked; if much reduced, practically no crepitation is heard.

Thread Test for Gastric Acidity.—A solid gelatin capsule containing a heavy powder (sulphate of sodium) giving a neutral reaction can be used. The capsule is pierced and a thread drawn through it which has been soaked for half an hour in a 0.25-per-cent. aqueous solution of Congo red. The thread is 120 centimeters long and, after passing through the capsule, is tied over it. The capsule is swallowed half an hour after a test breakfast, the free end of the thread being held in the hand. After fifteen minutes the thread is drawn out, the capsule having dissolved in the stomach in the

meanwhile. The end of the thread which has been in the stomach is now dark blue or violet according to the amount of hydrochloric acid in the stomach contents; if the thread is still red, this shows anacidity or that the capsule has stuck somewhere on its way. By this simple means the condition in regard to acidity in the stomach can be determined without inconveniencing the patient.

Friedrich's Test.—Another method of testing for the presence of hydrochloric acid, without the use of the stomach tube, is to color two threads Congo red, one a deeper tint than the other, tie them snugly to a small Einhorn stomach bucket, to which the usual long silk thread is attached for removal (see page 72), and have the patient swallow bucket and colored threads half an hour after a test breakfast, to be withdrawn a half-hour later. Alteration of the red color to blue indicates the presence of hydrochloric acid. Any device, such as tying a knot or a double knot in one of the threads, or making one shorter than the other, enables the observer to distinguish between the two original reds. A small metal button can be used instead of a stomach bucket, being placed within a gelatin capsule to prevent the saliva from coming in contact with the thread. Friedrich has given the name "Gastrognost" to this procedure.

Another Simple Quantitative Test of Acidity.—Take two strips of gauze, four or five centimeters long by one centimeter wide. Dip one strip in a 0.5-per-cent. solution of Congo red and the other in a 0.5-per-cent. solution of dimethylamidazo benzol. Fasten a long thread (70 cm.) to the two strips and pack the strips into a gelatin capsule, pass the free end of the thread through the cap of the capsule with the aid of a needle, then close the capsule with the cap and have the patient swallow it an hour after a test breakfast. When the capsule has dissolved, the strips of gauze are to be drawn out by means of the thread, and the tint of the strips will indicate the acidity of the stomach contents.

Gönsburg's Method of Testing the Absorptive Power of the Stomach.

Two centigrams of potassium iodid are placed in a section of very thin though strongly vulcanized rubber tubing about three-quarters of an inch in length. The ends of the tubing are folded and tied with threads of fibrin hardened in alcohol. To make sure that both ends are water-tight, the tube should be placed in water and allowed to remain for several hours, the water being then tested for potassium iodid. Should none of the drug be found, the patient is directed to swallow the package three-quarters of an hour after having partaken of an Ewald test meal. Free hydrochloric acid of the stomach will dissolve the fibrin threads and liberate the potassium salt into the stomach. The saliva should be tested for potassium iodid at intervals of fifteen minutes. When the acid secretion is below normal the salivary reaction will be delayed. In cases in which the acid secretion is wholly absent the potassium salt may not appear in the saliva for at least six hours.

Sahl's Desmoid Test. By the simple means of investigation of the functions of the stomach described by Sahl the physician may avoid the annoyance that certain patients experience when gastric contents are withdrawn by means of the stomach tube for analysis. The desmoid test is based upon the observation of Adolf Schmidt, that the digestion of raw connective tissue is confined to the stomach. Raw connective tissue passing through the stomach undigested is not affected by the pancreatic and intestinal juices, but is ejected with the feces unchanged. The details of the test are as follows: Two small squares of rubber dam, such as dentists use, are made into bags; into one is placed 1 decigram of iodoform and into the other 5 centigrams of methylene blue. The bags are closed and tied tightly with No. 00 raw catgut that has been allowed to dry but has not been treated chemically. The patient is instructed to swallow the two rubber bags with their contents. Under normal conditions of gastric secretion the catgut is duly dissolved and the contents of the bags liberated into the stomach; iodine will therefore shortly appear in the saliva, and methylene blue in the urine. Beginning three hours after the bags are swallowed, the urine and the saliva should be tested at one-hour intervals. Should the rubber bags with their contents pass through the digestive canal unchanged, gastric secretion is either very much retarded or entirely absent; in such cases no change is detected in the saliva or urine. The best time for making the test is immediately after the noon meal. Under normal conditions iodine will appear in the saliva in about two hours and methylene blue in the urine within six hours. Any deviation from this indicates hyperacidity or subacidity, according to the interval between the administration of the test agents and their presence in the saliva or urine.

MOTOR FUNCTION OF THE STOMACH.

The motor function of the stomach may be determined by the introduction of food and subsequent examination of the stomach contents. For this purpose Leube's test meal is employed, which consists of a plate of soup, beefsteak, a roll, and a glass of water; or Riegel's, consisting of 300 Cc. of beef broth, 180 grams of beefsteak, 60 grams of mashed potatoes, and one roll. After this meal the patient must not partake of anything during the next seven hours. At the end of the seven-hour period the stomach is washed out, the funnel being twice filled with about a half liter of water. If no food remnants appear, it may be concluded that the motor function of the stomach is normal.

For practical purposes the motor function of the stomach may be determined by means of a test breakfast. Under normal conditions the test breakfast leaves the stomach in two hours at most; so if at the end of two hours large quantities of fluid or food remain

nants are present, the motor function of the stomach may be regarded as impaired.

Chlorophyl Test.—A simple and rapid test for gastric motility is the chlorophyl test of Boas. In the morning, on an empty stomach, the patient drinks 500 Cc. (1 pint) of water to which 1 Cc. (16 minims) of a strong aqueous solution of chlorophyl has been added. When motility is normal, all but about 60 Cc. (2 ounces) of the water passes through the pylorus within half an hour. The liquid contents of the stomach can then be removed with a stomach tube, and the recovered chlorophyl, a substance which is not absorbable by the gastric mucosa, enables us to estimate the amount of water remaining of the 500 Cc. administered. Not only the fact but the degree of unpaired motility is indicated by the liquid residue above 60 Cc. (2 ounces).

PERMEABILITY OF THE PYLORUS.

Einhorn has described a method for testing the permeability of the pylorus. The patient is instructed to swallow beads filled with methylene blue and coated with mutton tallow. Inasmuch as fat is dissolved in the duodenum, a green or blue colored urine would indicate that the bead had passed the pylorus and that its contents had been absorbed. Under normal conditions the bead will pass into the duodenum, the tallow coating be dissolved, and the methylene blue appear in the urine in three to five hours.

The duodenal bucket devised by Einhorn (Fig. 8) is much smaller than the stomach bucket. It is fastened to the end of a braided silk thread about 80 centimeters in length, and is administered to the patient in a gelatin capsule an hour after a small meal. The bucket should be left in the intestinal canal three hours, during which time the patient should not partake of any food. The thread at its free extremity should be tied to the ear so that it cannot go beyond the 75-centimeter mark. After the expiration of three hours the bucket is slowly withdrawn. The resistance offered at the esophageal entrance at the larynx can be overcome by the patient going through the act of swallowing. It is better that the patient swallow the bucket before retiring at night and that it be withdrawn in the morning while the stomach is empty. If the bucket has entered the duodenum, its contents will be found to be yellowish, from bile.



FIG. 8. Duodenal bucket.

We can assure ourselves of the presence of the bucket in the duodenum by means of the Roentgen ray.

MICROSCOPIC EXAMINATION OF STOMACH CONTENTS.

Microscopic examination may be made of the gastric contents as withdrawn from the stomach by the stomach tube after the administration of a test meal, or from the vomitus. Undue importance should not be attached to the presence of meat shreds (Fig. 10, *C*) or starch granules (Fig. 9, *C*), which are practically never absent from the gastric juice. Normal gastric juice may also contain small particles of mucus, a few bacilli, and some yeast cells.

In motor insufficiency, remains of food which has been introduced many hours previously may be found in the form of numerous fat globules or fatty acid crystals (Fig. 9, *B* and *E*), vegetable fibers and plant cells (Fig. 9, *D*), as well as a few red blood-corpuscles which have come from abrasion of the pharynx by the stomach tube. Any red blood-cells found are apt to be altered in appearance as a result of the action of the hydrochloric acid of the stomach.

The Boas-Oppler bacillus (Fig. 10, *B*) is found in 75 to 85 per cent. of all cases of gastric carcinoma and rarely in non-malignant disease. It is found more frequently when lactic acid is present in large amounts, and may be absent in the incipient stages of carcinoma. It is 3 to 10 microns in length and 1 micron broad. These bacilli are frequently found joined end to end, forming very long chains. They stain by the ordinary method as well as by Gram's method, and take on a brown color when treated with iodine. This latter feature distinguishes them from the *Leptothrix buccalis*, which stains blue with iodine. The Boas-Oppler bacillus is not infallibly pathognomonic of carcinoma; it is present on rare occasions in the dilatation of benign stenosis of the pylorus.

Sarcinae are occasionally found in normal gastric juice, and especially in cases of gastric dilatation when there is marked fermentation, with hydrochloric acid present; they consist of cocci arranged in squares or tetrahedra (Fig. 9, *F*), and are of no pathologic significance other than being indicative of stagnation. A large number of yeast cells are found along with the sarcinae.

Mould fungi are occasionally found in the stomach contents, though in the normal stomach they are scarcely to be detected, since they mix at once with the chyme and are carried onward through the pylorus. Should a colony of the fungi infest a fold of the surface of the gastric mucous membrane, they may become so firmly adherent as to grow there undisturbed. When found, lavage of the stomach will remove the mould flakes. The mucous membrane is not directly injured by mould fungi.

Protozoa have been found in the gastric contents. Flagellates,

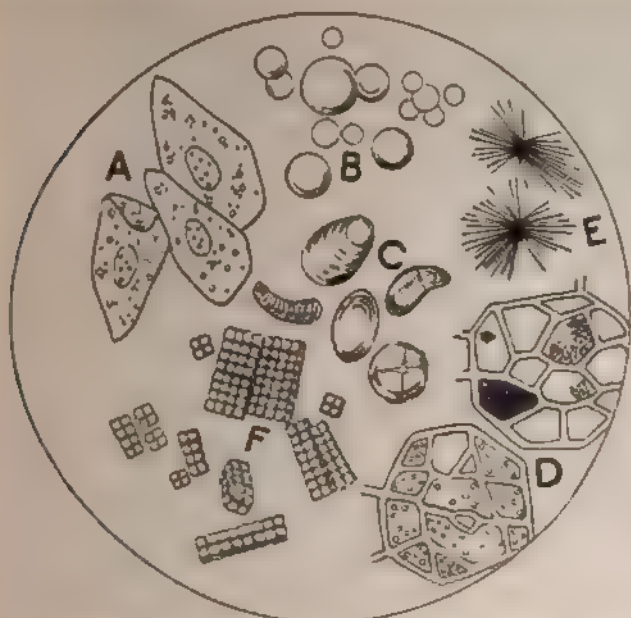


FIG. 9.—A, epithelial cells B, fat globules, C, starch granules, D, plant cells; E, fatty crystals F, sarcosine.

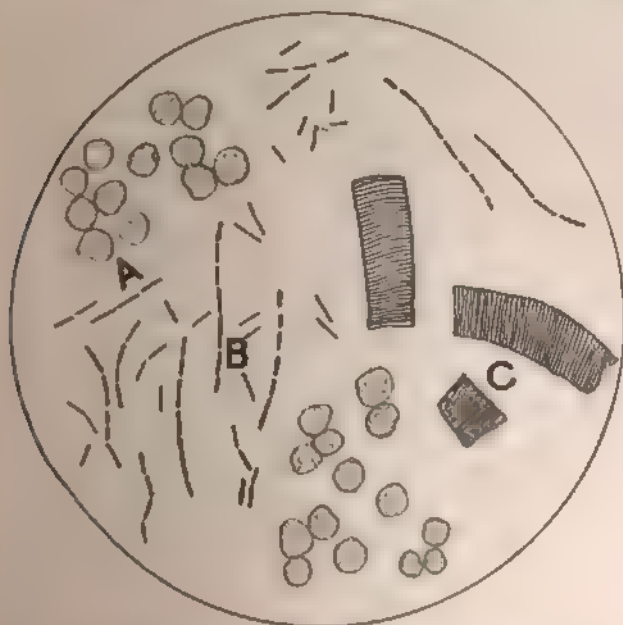


FIG. 10 —A, pus cells, B, Boas-Oppler bacilli, C, muscle fiber.

amebæ and monads are among the more frequent protozoan types found. According to Simon, "from the available data there can be no question that the presence of protozoa in the stomach contents is suggestive of non-obstructive carcinoma."

In cases of chronic gastritis, ulcer, hyperchlorhydria, and especially carcinoma, small shreds of mucous membrane are sometimes found in the gastric contents withdrawn by the tube. Such tissue fragments should be carefully studied under the microscope, since it is sometimes possible to make a diagnosis of carcinoma thereby.

Various types of crystals are occasionally noted in the gastric contents, among which may be mentioned bile acids, cholesterol, fatty acids, leucin, tyrosin, and calcium oxalate.

CHANGES IN GASTRIC SECRETION DUE TO PATHOLOGIC CONDITIONS.

1. **Gastric Neuroses.** The gastric findings in nervous dyspepsia show the acidity to be normal or either above or below; the ferments are fairly constant. The fact that the acidity varies from day to day, being one day excessive and the next decreased, is characteristic of the disease. Hemmeter gave the name "heterochylia" to this condition. In chronic gastritis the acidity remains constant, while in nervous dyspepsia it is subject to variation. The ferments, which are diminished in chronic gastritis, are usually normal in nervous dyspepsia. The findings in chronic gastritis reveal much mucus, in nervous dyspepsia little or none. The former condition is associated with dietetic errors, the latter with a neurotic temperament.

2. **Hyperacidity; Hyperchlorhydria.**—This term is used to designate the secretion of gastric juice of excessive acidity, the amount of free hydrochloric acid varying from a small to a high degree above the normal. The normal acidity is between 40 and 60 degrees. Usually an increased total acidity is found along with the increase in free hydrochloric acid. Hyperacidity is said to exist when there is a constant of more than 60 degrees, 0.2 per cent. of hydrochloric acid. Hyperacidity or hyperchlorhydria may be due to neuroses, or to pathologic changes in the mucous membrane of the stomach itself. Often there is diminished motility due to pylorospasm, and as a result stagnation of gastric contents with fermentation. In such cases the acidity may amount to 150 degrees or over; erythrodestrin is present in large quantities.

3. **Hypersecretion; Gastrosuccorhea; Gastrorrhæa; Gastrochylorrhæa.**

By this is understood an excessive secretion of gastric juice in the total or almost total absence of stimulus to the secretory function of the stomach. Hypersecretion, or gastrosuccorhea as it has been called, is always a pathologic condition. The diagnosis, as stated elsewhere, is confirmed by the finding of a pathognomonic quantity

of gastric juice, containing both hydrochloric acid and pepsin, in the fasting stomach. The quantity should be at least 140 Cc. before the clinician is justified in making a diagnosis of hypersecretion. There is somewhat of an increase in the degree of acidity; erythro-dextrin and achroodextrin are absent. There must be no food remnants, sarcine, or yeast cells. In gastric dilatation which may result from spasm of the pylorus we find fermentation products, yeast cells, and sarcine.

4. **Acute Gastritis.** Examination of the gastric contents in this condition reveals a diminished total acidity with little or no free hydrochloric acid. The total acidity is always below 40 degrees. Much mucus and undigested food is apt to be found. The hydrochloric acid secretion is either very much diminished or entirely absent.

5. **Chronic Gastritis.** Examination of the stomach contents in this condition reveals much mucus usually mixed with the food, which shows little signs of digestion. The quantity varies from 100 to 200 Cc. Free hydrochloric acid is diminished or absent, and the gastric ferments are very much reduced. The total acidity is below 40 degrees. Pepsinogen and rennin zymogen are always present. Erythro-dextrin is found in small quantities, and achroodextrin in abundance. The presence of epithelial cells and leukocytes is detected by microscopic examination. The finding of large amounts of mucus in which are mingled leukocytes and epithelial cells is characteristic of chronic gastritis.

6. **Achylia Gastrica.**—For a diagnosis of this condition the Ewald-Blois test breakfast may be used with advantage. Examination of the stomach contents shows very little change in the ingested food. There is usually a small amount of fluid present. The food has a characteristic appearance, showing complete lack of digestion. There is no free hydrochloric acid, and the total acidity is very low, 1 to 6 degrees. The gastric ferments are either very much diminished or entirely absent. There is no evidence of decomposition, no odor, and no mucus. Erythro-dextrin is absent. Lactic acid is present in very minute quantity if at all.

7. **Motor Insufficiency (Atony and Dilatation).**—When motor insufficiency is suspected, a tablespoonful of currants should be given to the patient in the evening, to be followed by a test breakfast the next morning (see page 67). Boiled rice may be given instead of currants. If either the currants or the boiled rice, as the case may be, be found in the fasting stomach next morning or removed with the test breakfast, a diagnosis of motor insufficiency is made. The volume of the gastric contents is usually increased, so that, as a rule, more than 180 Cc. is found after the test breakfast. If after a full meal in the evening visible food remnants are found in the fasting stomach in the morning, in all probability the condition is one of motor insufficiency of the second degree, inasmuch as food remnants

are never found in simple atony. The quantity of residue found in the stomach is an indication of the motor power of that organ. In aggravated cases of motor insufficiency food residues are often found in the stomach seven hours after the administration of a test meal, when the stomach under normal conditions would be empty. In severe cases the quantity of urine excreted during the twenty-four hours is markedly diminished, whereas in atony or motor insufficiency of the first degree it is normal. The chlorophyl test shows larger quantities of water residue than when motility is normal. Owing to the variability in the gastric secretion in motor insufficiency, chemical analysis affords but little aid to the diagnosis. In the initial stages of gastric atony the secreting glands produce an excessive amount of gastric juice, followed by a diminution due to fatigue of the glands. At first the hydrochloric acid may show a marked increase, or it may remain normal for a long time. Some cases of motor insufficiency may, upon examination of the gastric contents, show subacidity or anacidity.

8. Pyloric Stenosis.—In this condition there is always retention of food in the stomach. Should the patient partake of mixed diet in the evening, and the gastric contents be removed the following morning, the various food residues can be recognized macroscopically. Dilatation of the stomach always accompanies pyloric stenosis. When the obstruction is of benign origin, free hydrochloric acid is usually present, whereas it is usually absent in cases of malignant origin. Lactic acid, which is absent in cases of benign obstruction, is usually found in malignant obstruction. In malignancy there is a marked decrease in total acidity of the gastric juice, while in benign obstruction the acidity may be increased several degrees. Rennin, always found in cases of benign stenosis of the pylorus, is frequently absent in malignant obstruction. The odor of the gastric contents is more marked and fetid in malignant than in benign stenosis. In the former condition the Boas-Oppler bacillus is found, while in benign cases it is absent. Sarcinæ, which may be present in benign stenosis, are usually absent in the malignant form. In pyloric stenosis the gastric contents, if withdrawn and allowed to stand in a glass, will separate out so as to form three layers or strata. The upper layer is frothy, due to decomposition; the middle layer is clear or slightly cloudy; the lowest layer is semisolid.

9. Pyloric Insufficiency.—The diagnosis of this condition is confirmed when the stomach is found empty after the administration of the Ewald-Boas test breakfast. The degree of pyloric insufficiency is ascertained by administering test meals on successive days and removing the contents at stated intervals, such as three-quarters of an hour, half an hour, and fifteen minutes, after the ingestion of the test meal. Chemical analysis of the gastric secretion may reveal the presence of hydrochloric acid, pepsin, and rennin, or these may be absent. The ready passage of air from the stomach

tube through the stomach into the duodenum points to insufficiency of the pylorus.

10. **Gastric Ulcer.**—The clinical symptoms are of greater importance than examination of gastric contents in the diagnosis of this condition. The use of the stomach tube is obviously inadvisable when ulceration is suspected. The vomitus consists of well-digested food, which may or may not be free from blood. If blood be present, it will be either of a fresh red color or dark. The total acidity, of which free hydrochloric acid constitutes the major portion, is usually increased; at times it may be three times the normal—up to 180 degrees. The test for occult blood will usually reveal it in the feces. (Plate IX.)

11. **Erosions of the Stomach.**—In this condition examination of the returned water from gastric lavage reveals small fragments of mucous membrane which, under the microscope, show blood corpuscles and gastric glands, the form of which is apt to be well preserved and distinct. These fragments of gastric mucosa are constantly found when the patient's stomach is washed out in the fasting condition (Einhorn). In perhaps the majority of cases of gastric erosion there is a decrease in the hydrochloric acid secretion. On rare occasions, on the other hand, hyperacidity may exist. Mucus is present in greater or less quantity.

12. **Gastric Carcinoma.**—In this condition examination of the stomach contents yields certain results suggestive of the disease. Among these is the absence of free hydrochloric acid. This is among the early symptoms in perhaps 90 per cent. of all cases, but is subject to marked variations from day to day. The total acidity, as well as the amount of free hydrochloric acid, is diminished. Free hydrochloric acid may, however, be present in normal or more than normal amounts when the carcinoma is small and ulcerous and occupies the pyloric region.

The presence of lactic acid, increased in amount, is also suggestive of carcinoma of the stomach; 90 per cent. of cases show lactic acid present as well as free hydrochloric acid absent. In testing for lactic acid the contents of the fasting stomach should be examined in the morning, after thorough gastric lavage the night before. In carcinomatous cases this examination will show very slight digestion of proteins, with fairly good digestion of carbohydrates. The finding of aminoacids in the gastric contents is important. The microscope may show fragments of the neoplasm, such as cellular masses, embedded in blood—a very definite diagnostic sign. The cytodiagnosis is very helpful. The Boas-Oppler bacillus (Fig. 10, B) is said to occur in 75 to 85 per cent. of carcinomatous patients and is rarely found in any other. The test for occult blood in the feces is usually positive. The Wolff-Jungmans test is very valuable in conditions of achlorhydria (see page 87). Gluzinski's test is usually positive (see page 86).

CHAPTER III.

EXAMINATION OF THE DUODENAL CONTENTS.

WITH the discovery that the contents of the stomach could be removed at any stage in the process of digestion, by inserting a rubber tube and applying suction or the siphon principle, the diagnosis of diseases of the stomach by examination of its contents assumed an importance of the first rank. Then, as the stomach tube demonstrated its value, the question arose: Why not also examine the secretions of other organs concerned in digestion?—particularly the pancreas, which was known to be of much greater importance in the process of digestion than the stomach.

In 1897 Hemmeter described an apparatus by which he was able to introduce instruments into the duodenum. It consisted of a tube connected with a rubber bag the shape of the stomach, grooved on its upper surface for the passage of another tube into the duodenum through the pylorus after the bag had been introduced and filled with air. The difficulty with this instrument is that stomachs vary in size and configuration. One cannot obtain in advance a mold of any particular stomach so that the bag will tally exactly with its shape, and therefore it is impossible to say beforehand whether the exit of the groove in the bag is opposite the pylorus or not.

One year later Kuhn endeavored to reach the duodenum directly by means of specially constructed tubes with a metal spiral inside. The spiral served to prevent too much bending and kinking under pressure. He took a long stomach tube, put the metal spiral inside, and manipulated it for a long time, until he reached the duodenum. Kuhn's tube did not differ much from the ordinary stomach tube and was never practical.

Einhorn Duodenal Tube.—The credit for aspirating the duodenal contents is due to Einhorn, who in 1909 employed a small rubber tube ending in a perforated metal capsule (Fig. 11, *a*). For aspirating the duodenal contents this device is more practical than the duodenal bucket (see page 91). The main principle of the Einhorn duodenal tube is to allow the stomach to do the work of pushing the tube through the pylorus. The terminal capsule is 14 millimeters long and 23 millimeters in circumference, and can be unscrewed for cleansing. It communicates with a long, thin rubber tube (8 millimeters in circumference and one meter long), marked at 40 cm. (I, cardia), 56 cm. (II, pylorus), 70 cm. (III), and 80

cm. distant from the capsule. At the free end of the tube is a tip to which a syringe can be easily attached (Fig. 83). This apparatus can be used for aspirating either gastric or duodenal contents. For the latter purpose it is introduced in the same manner as for

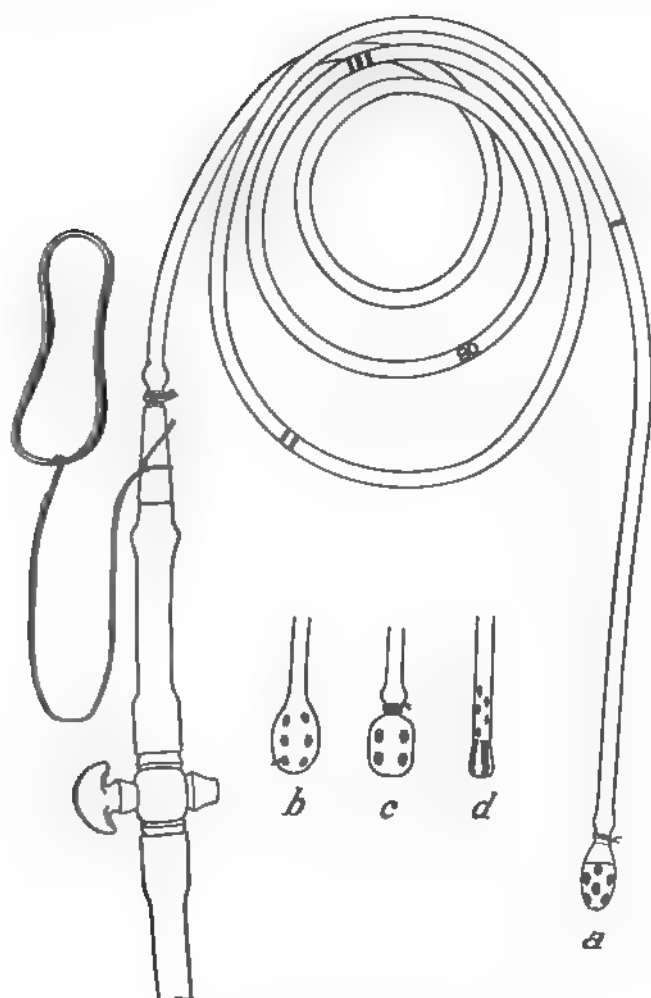


FIG. 11.—*a*, Einhorn duodenal tube; *b*, Gross duodenal tube; *c*, Palefski duodenal tube; *d*, Jutte duodenal tube.

duodenal feeding, as described on page 500. After the syringe is filled with the duodenal contents, the tube is clamped and slowly withdrawn. When the capsule reaches the level of the cricoid cartilage the patient is told to swallow, and during this act the capsule is withdrawn. (Plate V.)

Gross Duodenal Tube.—The Gross duodenal tube is somewhat similar to Einhorn's, except that it has a multiperforated silver-plated leaden ball weighing 10 grams at its distal end, covered by the tube on all sides (Fig. 11, *b*). This heavy end facilitates the passage of the tube through the pylorus. In detail the technic of introducing the Gross tube is as follows: The fasting patient sits in a chair and with the aid of a little water swallows the metal end of the tube; then, breathing rhythmically, he simply allows the tube to pass in up to the 45-centimeter mark; then he holds it fast with his lips, bends forward a moment, and lies on his right side, with raised upper body. The tube is then held lightly between the fingers and is fed into the stomach up to the 70-centimeter mark; it is not shoved in, but allowed to follow the pull of the heavy metal end and of the respiratory movements. Then gastric juice is aspirated and tested for its reaction. The patient remains five minutes longer on the right side, with the tube held fast between the lips; he then rolls over on his back, and two pillows are placed under his hips. Five minutes later the tube is inserted to the 80-centimeter mark. After another five minutes (often earlier) the aspirated fluid is neutral or alkaline and thus found to be from the duodenum. To aid the passage of the duodenal tube, percussion of the fifth dorsal vertebra is helpful (see page 211). This pyloric reflex opens the pylorus, while the stomach forces the weighted end of the tube into the duodenum.

Palefski Duodenal Tube.—The Palefski tube has a gold-plated leaden ball at its distal end (Fig. 11, *c*), and, like the Gross tube, passes into the stomach and through the pylorus by the force of gravity.

Jutte Duodenal Tube. The Jutte tube has a lead sinker at its distal end, and perforations in the soft-rubber tube above it (Fig. 11, *d*). A wire obturator assists in the rapid passage of the tube, which is accomplished in much the same manner as the passage of an ordinary stomach tube.

Determination of Tube in Duodenum.—To determine whether the tube is in the stomach or the duodenum, aspirate, and if you find that the fluid comes out quickly and looks watery, that hydrochloric acid is present, that Congo paper turns blue, dimethyl-amidoazobenzol red, and litmus paper red, you know the tube is in the stomach; if, on the contrary, you get only very little fluid, of neutral or alkaline reaction and golden yellow color, you know it is in the duodenum. Sometimes a patient has no hydrochloric acid in the gastric contents, and the achlorhydria may lead to the inference that the tube is in the duodenum. Under such circumstances have the patient drink some milk, and aspirate; if the milk is not obtainable immediately after drinking, it will be known that the tube is in the duodenum. Conclusive proof is afforded by drawing the tube up a short distance and again aspirating, when,

PLATE IV



Duodenal Tube in the Duodenum, with Perforated Metal Capsule Near the Papilla of Vater.

the terminal capsule having re-entered the stomach, you will obtain the milk that the patient has just swallowed. If the patient has had milk before the test, substitute raspberry syrup as an indicator. If necessary, a Roentgen-ray inspection can be made.

Characteristics of Duodenal Contents. The duodenal contents usually consist of pancreatic juice, bile, and some duodenal secretion proper. The mixture normally has a golden yellow hue, is limpid, viscid, and alkaline to litmus and methyl orange (alkalinity = about 20), but rather acid to phenolphthalein. Ordinarily, after standing a few hours, the yellow color loses much of its brightness and becomes slightly greenish and turbid. The fresh contents have no particular odor, but with the change in appearance an unpleasant odor develops, which rapidly increases in intensity; this result is due to decomposition, or rather putrefaction, through the action of bacteria. Finally the odor becomes fetid, resembling feces.

Urobilin and Urobilinogen.—The excretion of urobilin and urobilinogen in the duodenal contents is definitely increased in hemolytic anemias. Schlesinger's method of testing for these substances is as follows: To 10 Cc. of the duodenal contents an equal amount of a saturated alcoholic solution of zinc acetate is added. After being thoroughly mixed by vigorous shaking, the whole is filtered through coarse filter paper. To 10 Cc. of this filtrate, 1 Cc. of Ehrlich's aldehyde reagent is added with a pipet. If urobilinogen is present the fluid becomes cherry red, and when there is a preponderance of urobilin the color by transmitted light is yellow or brown. The liquid is allowed to stand fifteen minutes and is then examined spectroscopically. Urobilin is marked by a broad band in the blue end, while urobilinogen absorbs a narrow portion of the spectrum in the yellow at the edge of the green and if present in large amounts may obliterate the entire yellow portion of the spectrum.

Test Meal.—Einhorn has devised a test meal for use in connection with the examination of the duodenal contents, similar to the test breakfast for examination of the gastric juice. Inasmuch as the duodenal tube has a narrow lumen, solid food would clog up the instrument and is therefore inappropriate for such a test. The test meal consists of one bouillon cube to one cup of hot water. Bouillon contains the beef extractives which act as a strong stimulant to the digestive secretions. In all pancreatic affections and other digestive disturbances in the duodenum it is best to examine the duodenal contents from one to one and a half hours after the bouillon test meal. The pancreatic secretions are at their best in an alkaline medium, and there should be no acid gastric contents in the duodenum when the duodenal contents are withdrawn. In pathologic lesions we may have conditions in which the degree of alkalinity is too high or too low.

For the diagnosis of liver and gall-bladder lesions it is better to

examine the duodenal contents while the patient is fasting— that is to say, without a test meal. The bile is then in as pure a state as we can obtain it for this purpose.

Examination for Enzymes.—Besides testing the duodenal contents for reaction (neutral, acid, or alkaline), specific gravity, appearance, and admixture of mucus, the principal point of importance is to ascertain the presence or absence of the three pancreatic ferments, amyllopsin, steapsin, and trypsin, and if possible the approximate quantity of each. Einhorn with his agar tubes (Plate IV) has made it possible for us to follow one uniform method of examination, so that comparisons may be easily made and the results accurately tabulated.

Einhorn's agar tubes are made as follows:

Starch Agar Tubes.—Agar powder, 2.5 Gm.; starch, 5 Gm.; distilled water, to 100 Cc. Rub the starch and agar in a mortar with sufficient water to make a smooth paste, then add the balance of the water. This mixture is put into a flask and heated to the boiling-point. It is then drawn by suction into a capillary glass tube (inside diameter, 1.5 millimeters) which has previously been warmed in the flame. The tube and contents are allowed to cool and are then cut into 3-centimeter lengths. These are sealed at each end with melted paraffin. (Plate IV, A.)

Olive Oil Agar Tubes.—Olive oil, 25 Cc.; agar powder, 2 Gm.; aqueous solution of Nile-blue sulphate (1:2000), sufficient to make 100 Cc. Rub the olive oil and agar together, add sufficient water to make a thin paste, and add the Nile-blue sulphate solution up to 100 Cc. Then proceed the same as with the starch tubes. (Plate IV, B.)

Hemoglobin Agar Tubes.—Hemoglobin powder, 1 Gm.; agar powder, 2.5 Gm.; distilled water, sufficient to make 100 Cc. Rub the hemoglobin with about 10 Cc. of water until it is smooth; add the agar powder and the balance of the water; then proceed in the same manner as with the starch tubes. (Plate IV, C.)

The agar tubes are best kept on ice until used. They retain their efficiency for a month or six weeks, when they begin to deteriorate. When they become dry they are apt to give misleading results.

Mode of Procedure.—Take one tube of each kind, starch, oil and hemoglobin, scrape off the paraffin at one end, and place the tubes vertically, denuded end down, into a bottle containing a small quantity of the duodenal secretion (Plate IV, D). Add a few drops of toluol, and keep the bottle in a thermostat at blood temperature for sixteen to twenty-four hours. (Einhorn uses a Freas electric oven.) At the end of this time the tubes are taken out of the bottle, wiped off, and inspected. The starch column is colorless as far upward as the amyllopsin has acted upon it, converting it into sugar; the distance is recorded in millimeters

PLATE V



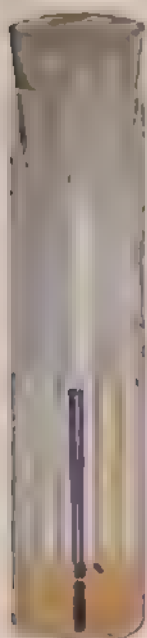
A



B



C



D



E



F



G

Agar Tubes for Testing Duodenal Contents

A - 10 cc. of agar tube. *B* - 10 cc. of agar tube. *C* - hemoglobin agar tube. *D* - bottle containing duodenal contents and agar tubes ready for the thermostat. *E* - after incubation of 10 cc. of agar and 5 cc. of duodenal contents into red solution. *F* - after incubation of 10 cc. of agar and 5 cc. of duodenal contents into red solution. *G* - after incubation of 10 cc. of agar and 5 cc. of duodenal contents into red solution.

•

•

(Plate IV, E). The starch column is further examined by pushing it out and dipping it into a weak iodine solution. The hemoglobin tube shows a change of appearance (due to the action of the trypsin), the lower end having become more or less transparent (Plate IV, G). The oil tube has a bluish appearance at the lower end, the steapsin having split the oil into fatty acids producing this color (Plate IV, F). The extent of the transparency in the hemoglobin tube and of bluish color in the oil tube are measured and stated in millimeters. Thus the starch serves to gauge the amyllopsin, the olive oil the steapsin, and the hemoglobin the trypsin ferments.

Determination of Enzymes.—The duodenal ferments in normal individuals, as determined by Einhorn and measured in millimeters on the capillary tube (Fig. 12), fluctuate as follows: Amylopsin, 4 to 8 mm.; steapsin, 2 to 5 mm.; trypsin, 0.5 to 5 mm. The average figures are: Amylopsin, 6 mm. (Plate IV, E); steapsin, 3.5 mm. (Plate IV, F); trypsin, 2.8 mm. (Plate IV, G).

Changes in Pathologic Conditions.—In pathologic conditions there is a noticeable independence among the three different ferments with regard to quantity in the same individual: one ferment may be present in large amount and the other two in small amount or not at all. It is therefore necessary to test for each of the three ferments separately. This is accomplished most conveniently by means of the agar tubes (Plate IV).

The pancreatic secretion is subject, like the gastric juice, to functional anomalies, or deviations from the normal. It may contain an overabundance of ferments or too small a quantity. While in the case of the gastric juice the functional activity is usually reckoned by the amount of hydrochloric acid present, we have not been able as yet to select any one of the three pancreatic ferments for reckoning purposes. However, trypsin being the most important ingredient of the pancreatic juice, it would seem natural to use it as a gauge for the functional activity of this gland. According to Einhorn, the following terms may be advantageously used:

Eupancreatism: Normal function; all three ferments present, trypsin showing the normal quantity (1 to 4 millimeters).

Hyperpancreatism: Increased activity; all three ferments present, trypsin existing in excess (above 4 millimeters).

Hypopancreatism: Diminished activity; the three ferments present, trypsin decreased (below 1 millimeter).

Dyspancreatism: Disturbed function; one or two of the three ferments are absent.

Heteropancreatism: Varied function; the presence and amount of ferments showing no constancy, but varying from time to time.

In addition to these connotations on the pancreatic juice which

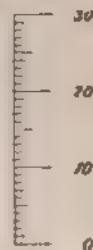


FIG. 12. Millimeter scale.

refer to degrees of ferment activity, we must distinguish differences in quantity also. There is, then, normal pancreatic secretion, or *euchylia pancreatica*; too much secretion, or *hyperchylia pancreatica*; and too little secretion, or *hypo-chylia pancreatica*. *Achylia pancreatica* signifies that there is no pancreatic juice at all.

Exact knowledge of the state of pancreatic activity in the patient will often assist, in conjunction with other symptoms, in the diagnosis of functional or organic disease of this gland. Moreover, it is always helpful in formulating an appropriate diet for any given case no matter from what disease the patient may be suffering.

Direct Medication.—The duodenal tube may be used for direct medical application to the mucous membrane of the upper intestinal tract. It has been found that instillation of medication into the duodenum will not only improve inflammatory processes present but also act beneficially on the mucous membrane of the common bile duct, gall bladder, and pancreas. Cholecystitis will often yield to instillation into the duodenum, every day, of 15 Cc. of either a 0.5-per-cent. solution of argyrol or a 1-per-cent. solution of ichthyol. A turbid bile means a probable cholecystitis. In the treatment of cholecystitis, direct application of solutions of argyrol and ichthyol can easily be made through the duodenal tube to the duodenum just above the ampulla of Vater.

Draining the Gall Bladder and Bile Ducts.—When surgeons find an inflammation of the gall bladder on removing gallstones, they are in the habit of draining the gall bladder. Favorable results are attained by cholecystostomy in cases of cholangitis, choledochitis, and cholecystitis. Meltzer found that a 25-per-cent. solution of magnesium sulphate poured directly on the mucous membrane of the duodenum caused complete local relaxation of the intestinal wall. It may relax the sphincter of the common duct sufficiently to permit the ejection of bile, or even the removal of a calculus of moderate size wedged in the duct in front of the papilla of Vater. Lyon¹ has clinically verified the scientific results of Meltzer, so that we may now not only relax the sphincter of the common bile duct and thereby drain all the ducts but also cause the gall bladder to be compressed and empty its contents (see Law of Contrary Innervation, page 65). It is thus possible to differentiate and study:

1. The first bile from the common duct.
2. The second, darker and more viscous bile, from the gall bladder.
3. The third, clear golden bile, from the hepatic duct.

We can thus make a differential diagnosis between cholecystitis and choledochitis.

After the tube is in the duodenum 100 Cc. of 25-per-cent. magnesium sulphate is allowed to flow by gravity into the duodenum. In two to ten minutes bile will be recovered which will be recognized

¹ B. B. Vincent Lyon: Can the Gall Bladder, Biliary Ducts and Liver be Medically Drained? *American Journal of the Medical Sciences* October, 1920.

DUODENAL LAVAGE

by its light yellow color. To quote from Lyon:¹ "This first I may be from 10 to 20 Cc. in amount and may require from one to three minutes to aspirate, when a sudden transition appears (seen first in the glass window of the tube) and the bile becomes darker, more viscid and more concentrated, and, in normal gall bladders, remains transparent but is more of a molasses yellow. We believe this type of bile to be that stored up in and delivered from the gall bladder, and when it appears first in the glass window the second sterile bottle is detached and replaced by a third sterile collecting bottle into which the bile is allowed to flow until all of this darker bile (more viscid, transparent or turbid) has been collected and is being replaced by a lighter yellow, thinner and usually transparent bile which is aspirated much more slowly and intermittently, and which we believe to be bile freshly secreted from the liver." It will thus be seen that in duodenal lavage with a solution of magnesium sulphate (see page 106) we have a method for the relief of biliary stasis, the forerunner of cholecystitis and cholelithiasis, and also a rational treatment for these diseases if they are present.

Oxygen Insufflation.—Oxygen insufflation has been found very useful as an intestinal antiseptic measure. Bacterial proliferation in the intestine can be inhibited by insufflation of pure oxygen through the duodenal tube into the duodenum. Schmidt has found this method of treatment in intestinal fermentative dyspepsia very satisfactory. Equally favorable results are obtained in cases of intestinal putrefaction. Oxygen exerts a directly destructive effect on all anaërobic bacteria; and when introduced into the intestine in the manner mentioned, there can be no dodging the issue. The technic of oxygen insufflation is described on page 679.

Duodenal Lavage.—Duodenal lavage was first practiced and described by M. Ernest Jutte, of New York. It is to the small intestine what an enema is to the colon. In applying duodenal lavage the apparatus employed must enable the practitioner to bridge the stomach, so that the irrigating fluid will not enter that organ but go directly into the duodenum, thus avoiding vomiting.

The outfit required consists of a thin rubber duodenal tube, an aspirating bottle, a suction-pump or an ordinary syringe, an irrigator and rubber connections; and if the patient finds it difficult to swallow the tube, a thin wire is fitted into it to serve as a guide (Fig. 133).

Duodenal lavage should be done when the patient's stomach is empty. The technic of the operation is as follows: The tube is first moistened, then either swallowed or introduced manually as far as the "III ring" mark; if the manual method is adopted, the wire obturator must first be pushed down inside the tube as far as

¹ B. H. Vincent Lyon, *Diagnosis and Treatment of Diseases of the Gall Bladder and Biliary Ducts*, Journal of American Medical Association, Sept. 27, 1910.

it will go—that is to say, to the lead sinker. The patient drinks a tumblerful of water and lies down on his right side. Gravity carries the heavy sinker near the pylorus, and peristalsis soon pushes it through along with the water—in the course of two or three minutes, usually. Connections are then made between tube and aspirating bottle, and between bottle and suction apparatus. Exhaust the air from the bottle with a large syringe; the suction will soon result in the appearance of diluted duodenal secretion in the aspirating bottle. Examination of this fluid makes it clear that the tube has passed through the pylorus into the duodenum (Plate V). The patient can now sit up. Disconnect the suction bottle and connect the tube with the container holding the irrigating fluid. Allow 500 to 1000 Cc. (1 to 2 pints) to trickle through the tube, taking fifteen minutes for this procedure. Should the patient complain of any discomfort, stop the drip for a while. The entire quantity of fluid will have reached the duodenum in ten to fifteen min-



FIG. 13.—Jutte's apparatus for duodenal lavage.

utes, peristalsis pushing it gradually onward so that there will be no distention in any part of the bowel. To make certain of this, the flow can be interrupted every few minutes. After the desired quantity of liquid has been introduced, the tube is gently withdrawn during respiration and the patient is allowed to rest.

Character of Irrigating Fluid.—Each case will determine the kind of fluid that is best to use. In nervous disorders, general malaise, anemia, rheumatism, indicanuria—in short, whenever it is desired to cleanse the bowel thoroughly—Jutte has found that a solution containing 9 grams each of sodium chlorid and sodium sulphate to 1000 Cc. (1 quart) of water passes through the bowel without being absorbed and acts excellently as an irrigant. Duodenal lavage thus flushes out the entire length of the intestinal canal from the pylorus to the rectum; it removes toxins of every nature, and restores by simple cleansing of the intestinal tract the local conditions necessary for normal health.

Plain distilled water induces a very copious diuresis, thus flush-

ing out the kidneys. In icterus and when fat digestion is impaired the addition of 0.5 Gm. ($7\frac{1}{2}$ grains) of pure Castile soap to 1000 Cc. (1 quart) of saline is beneficial. To withdraw fluid from the body tissues, a stronger solution than normal saline will be found necessary. By placing a thermometer against the tube near the patient's mouth the temperature of the fluid can be easily regulated so that it will enter the intestine at body heat. The bowels usually move freely within one or two hours after each duodenal lavage. When it is desired to drain the bile ducts and gall-bladder, 100 Cc. of a 25-per-cent. solution of magnesium sulphate should be employed (see page 695).

Indications for Duodenal Lavage.—Considering that so many diseases are caused by intestinal toxemia, Jutte believes that flushing out the toxins from the intestine gives good results in such cases as rheumatism, gout, sciatica, arthritis deformans, functional disorders of the heart, arteriosclerosis, nephritis, toxemia of pregnancy, cirrhosis of the liver, intestinal stasis, primary and secondary anemia, skin diseases, catarrhal inflammation of the mucous membranes, mucous colitis, asthma, pernicious anemia, neuroses, neuralgia, neuritis, insomnia, epilepsy, neurasthenia, melancholia, dementia and insanity.

In the practice of duodenal lavage I have not had a single failure as regards the clinical cure of intestinal stasis, and incidentally also of constipation, although the anatomical conditions (kinks and adhesions) remain unchanged. From this positive fact several obvious conclusions may be drawn. The first is that kinks and bands—which, indeed, have been pronounced physiologic by some authors—are not necessarily the cause of intestinal stasis, and that, consequently, their surgical removal will not cure the stasis. Another conclusion is that any other pathologic condition should disappear after successful duodenal lavage treatment, if really due to intestinal stasis; and if it does not disappear after the supposed causative factor has been removed, it follows that the etiology requires correction—that the condition was not, after all, due to intestinal stasis (see page 683).

BACTERIOLOGY OF THE DUODENUM

MacNeal and Chace have made a careful study of the bacteria in the duodenum with the aid of the duodenal tube. They found that the bacteria counted microscopically in the fluids from the duodenum varied from 600 to 860,000 per cubic millimeter, or from 600,000 to 860,000,000 per cubic centimeter. The bulk of these were dead, at any rate, the number brought to development in culture was only a small fraction of the number counted. The number of

bacteria which develop into colonies in cultures seems to bear a more definite relation to the gastro-intestinal condition. The fluids from which a million or more colonies per cubic centimeter developed were from patients suffering from various diseases, most of them very ill. We may conclude, therefore, that the number of colonies developed in cultures of duodenal fluid is, roughly, an index of the digestive derangement. When these are numerous, the acidity of the gastric juice is often diminished, or there is other evidence of abnormality in stomach or duodenum.

The duodenal fluid heated to 27° C. for fifteen minutes and then inoculated with spore material failed to develop colonies. This result suggests that the passage of bacterial spores through the stomach into the duodenum is not ordinarily a prominent factor in the bacterial involvement of the intestine.

The direct inoculation of fermentation tubes of glucose broth and lactose broth with the duodenal fluid, with subsequent incubation at 37° C., revealed gas-producing organisms in one-third of the fluids. These fluids were from patients suffering from influenzal pneumonia, typhoid fever, asthenic gastritis, gastric neurosis, delirium tremens, recurrent ulcer after gastroenterostomy, gastric ulcer, and atrophic gastritis. It would seem that gas production in these cultures takes place only when there is considerable disturbance of digestion. In some instances the gas was evidently produced by bacilli and in other instances by yeasts.

MacNeal and Chace conclude that the normal duodenal fluid during a fast is almost free from living microorganisms, although numerous bacterial cells are always visible on microscopic examination. The few living bacteria obtained in cultures from such fluids are mostly Gram-positive cocci. In various gastro-intestinal disturbances the number of cultivable bacteria in the duodenal fluid is markedly increased. These organisms are of several different varieties, bacilli, cocci, yeasts and branching thread forms being represented in different cases. In cases of typhoid fever the *Bacillus typhosus* can be isolated from the duodenal fluid. The bacteriologic study of intestinal juice obtained with the duodenal tube is of value in cases of achylia gastrica with diarrhea, and in cholecystitis, nor is it without promise in the investigation of those obscure diseases which are sometimes ascribed to abnormal intestinal digestion. Moreover, it may prove to be a valuable procedure in the early diagnosis of typhoid fever and in the detection of typhoid carriers. Cultures from duodenal contents removed by means of the duodenal tube are said to furnish a more reliable and simple method for the detection of typhoid bacilli than the stool examinations. The presence of pus and blood always indicates a pathologic condition. The microscope reveals epithelial cells, bile-stained cells, and mucus.

RESULTS OF THE DIRECT EXAMINATION OF THE DUODENAL CONTENTS

1. **Gall Bladder.**—The macroscopic appearance of the bile is of great diagnostic import. A clear, golden-yellow bile usually indicates a normal gall bladder. A turbid, greenish or dark brown bile, perhaps mixed with mucus, suggests a diseased state of either the gall bladder or the liver, or both. In gall-bladder affections the bile is liable to change in character; if turbid, this condition is due to admixtures acquired during its sojourn in the gall bladder. In chronic affections of the liver we can expect a fixed appearance of the bile, as its character is given it at the place of production.

2. **Cholecystitis.**—It is well understood that the diagnosis of cholecystitis or cholelithiasis cannot be made from the appearance of the bile alone. The latter, however, in conjunction with the other clinical signs, is of great assistance in establishing a correct diagnosis. In cases of cholecystitis the presence of innumerable bile-stained pus cells in the duodenal contents confirms the diagnosis. Turbid bile aspirated from the duodenum in the fasting condition is of importance in the diagnosis of chronic cholecystitis. Clear bile of a golden yellow color is never found in cases of cholecystitis. In some cases of cholecystitis, leukocytes are found in the duodenal contents; they are never found in duodenal contents from normal persons.

3. **Bile.**—Occasionally we obtain, on aspiration of the duodenal contents, at first only a clear or slightly amber-colored fluid, of alkaline reaction, containing the pancreatic ferments. Usually after waiting a short time, and after repeated aspirations, a golden-yellow fluid (containing bile) appears. This has no diagnostic significance. If, however, after patient waiting and aspirating, only pancreatic juice but no trace of bile appears, this fact may be of some importance, particularly in cases of chronic jaundice. If bile is present in the duodenal contents, complete occlusion of the common bile duct can be excluded. If the bile is entirely missing and pancreatic juice is present, it indicates that the seat of obstruction is above the common duct.

4. **Obstruction of the Common Duct.**—Absence of both bile and pancreatic juice suggests a mechanical obstruction of the common duct just above Vater's papilla, thus blocking the entrance into the duodenum of either bile or pancreatic juice. In these instances it is advisable to ascertain positively that the tube is in the duodenum, by the milk test or the Roentgen ray.

5. **Bile and Pancreatic Juice.**—Duodenal contents containing bile and pancreatic secretions permit gauging the pancreatic function.

6. **Pancreatitis.**—The presence of the three ferments in sufficient quantity indicates normal pancreatic activity. If one of the

ferments is constantly absent, the probability is that the patient has chronic pancreatitis. A tumor of the pancreas may, however exist, notwithstanding the presence of all three ferments. This surprising fact finds its explanation in the circumstances that the tumor has not invaded all the pancreatic tissue; enough remains unaffected to continue the pancreatic function undisturbed.

7. **Duodenitis.**—In duodenitis, mucus (stringy), Gram-positive motile bacilli, and numerous cocci are found.

8. **Duodenal Ulcer.**—In duodenal ulcer, blood and pus cells are frequently found in the duodenal contents.

9. **Typhoid Fever.**—In typhoid fever the specific bacilli are frequently present in the bile. Typhoid carriers can thus be easily detected.

10. **Pernicious Anemia.**—In pernicious anemia, urobilin and urobilinogen are found in the duodenal contents in far above normal quantity. Urobilinogen is increased when the anemia is severe. Following splenectomy there is a definite decrease in the amounts of urobilin and urobilinogen in the duodenal contents—especially a decrease in urobilinogen.

CHAPTER IV.

EXAMINATION OF THE FECES.

IN the various chapters treating of intestinal diseases and certain affections of the stomach (achylia, subacidity) it is pointed out that a systematic examination of the feces is of extreme importance for exact diagnosis. The best method is that of Adolf Schmidt; indeed, it may be said that Schmidt has established a functional intestinal diagnosis that is indispensable to the modern, up-to-date physician dealing with affections of the stomach, liver, gall bladder, and intestine.

The object of the functional diagnosis is to recognize disturbances in the physiologic action of the organs, notably at a time when gross objective signs are still absent or when there are either indistinct subjective sensations or no symptoms at all. This object is attempted in the demand made upon the organ for the performance of a selected task, and from the manner of its performance conclusions are drawn as to the capacity of the organ. In order to form a correct opinion on any functional disturbance, it is necessary to learn, by accurate and systematic observation, the normal capacity of the organ. By the modern method of introducing stomach tubes, it is possible to control to a nicety the performance of the stomach and to examine separately its three principal functions, motility, secretion, and absorption.

In intestinal pathology, unfortunately, we have as yet been unable to achieve the same measure of success in regard to functional diagnosis and the recognition of normal and pathologic function, as in gastric pathology. True, in the examination of intestinal function we are confronted with distinct difficulties that are not encountered in the examination of the stomach. In the latter organ we are dealing with a comparatively simple condition, there being only a question of the influence of two or three digestive secretions whose presence and effect upon the contents of a small and easily accessible space—the stomach—awaits examination. We are able to interrupt the digestive work of the stomach at a definite time and to ascertain a definite phase of intermediate digestion. In the intestine, however, the problem is totally different. Once the chyme has passed through the pylorus and arrived in the intestine, it is beyond our reach, and the best we can do in the presence of pathologic conditions is to exert an influence on the course of digestion and the time of retention of the chyme in the intestine. We are unable to interrupt at discretion the intestinal digestive process, and can only base

our judgment on the character of the feces as the end-product of total digestion in the gastro-intestinal canal—a process in which a great many ferments participate, the absence or insufficient functioning of which cannot always be determined with certainty.

Nothnagel was the first to evolve a valuable method of fecal examination, in the 'eighties. In later years his labors fell into neglect, and there were only sporadic contributions to the literature of fecal examination. The interest in these examinations had almost completely died out when Adolf Schmidt, as mentioned above, and his collaborator Strasburger, reclaimed coprology from oblivion, placing it on a theoretically and practically assured basis and rendering it accessible to wider circles.

The method of Schmidt and Strasburger consists in demanding from the intestine the performance of a certain task, and observing the manner in which it is performed on the principle of the test breakfast and test meal on which the function of the stomach is determined. The demand made upon the intestine is that it deal with a certain uniformly constituted diet, the so-called test diet. The question of whether and how the intestine digests and assimilates this test diet is determined by examination of the feces, so that the procedure of functional examination is divided into two parts: (1) administration of the test diet, and (2) examination of the stool resulting from the test diet.

THE TEST DIET AND ITS ADMINISTRATION.

The demands to be made upon a suitable test diet are many. It should be made up in such a way as to be equally acceptable to healthy and to intestinally diseased individuals. It should be almost, but not entirely, free from indigestible matter, so that the irritation normally supplied by the intestinal contents may not be entirely absent. Furthermore, it should satisfy the minimum caloric requirements in physical rest; it should contain a suitable proportion of protein, fat, and carbohydrates; it should be easily procurable and easy to prepare. In Schmidt's original test diet, importance was attached to the exact measurement of all the articles of nutrition contained. It was as follows:

Morning. Milk 0.5 liter, or, if milk is not well tolerated, cocoa 0.5 liter, prepared with 20 grams of powdered cocoa, 10 grams of sugar, 100 Cc. of water and 100 Cc. of milk; together with 50 grams of biscuits.

Forenoon. One-half liter of oatmeal gruel (oatmeal 40 grams, butter 10 grams, milk 200 Cc., water 300 Cc., one egg and a little salt, strained).

Midday. Chopped beef (125 grams gross weight) slightly roasted with 20 grams of butter, care being taken that the inside

remains rare. Mashed potatoes, 250 grams (potatoes 190 grams, milk 100 C.c., butter 10 grams, and a little salt).

Afternoon. Like the morning diet.

Evening. Like the forenoon diet.

This diet contains: milk 1.5 liters, two eggs, 100 grams of biscuits, oatmeal 800 grams, butter 50 grams, beef 125 grams, potatoes 190 grams, having the following composition:

	Protein	Fat	Carbohydrates
Milk, 1.5 liters	45.0	53.2	67.6
Two eggs	11.4	10.9	0.5
Biscuits, 100 grams	8.55	0.98	75.1
Oatmeal, 800 grams	1.76	1.2	8.2
Butter, 50 grams	0.47	42.2	
Beef, 125 grams	26.1	1.06	
Potatoes, 190 grams	3.95	0.28	39.9
	97.03	110.72	191.3

Calculating the protein at 4 calories, the fat at 9, the carbohydrates at 4, this test diet would yield 2131.8 gross calories. According to Lohrlich, the direct combustion of this test diet in the calorimeter yields 2146.3 calories, which corresponds closely enough. Also according to Lohrlich, the cellulose content of one day's test diet amounts to 0.8916 gram. However, in the course of years it has been found that, for practical purposes, the precise quantitative determination of the various nutritive ingredients is not at all necessary. It should simply be borne in mind that the test diet should be composed of milk in not too restricted quantities ($\frac{1}{2}$ to $1\frac{1}{2}$ liters; white bread or crackers, about 100 grams; potato purée, 100 to 250 grams; chopped beef, 120 grams. But many additions or omissions may be resorted to, to suit the taste and requirements of the patient. The accurately measured diet is now used only for exact clinical examination and quantitative analyses. For practical purposes Schmidt now lays down the following changed and amplified form of his test diet:

Morning. Milk $\frac{1}{2}$ liter, or tea or cocoa with much milk if acceptable, one roll and one soft-boiled egg.

Breakfast. Oatmeal gruel, strained, one plate, with a little salt or sugar if desired, farinaceous soup or porridge may be substituted.

Mid-day. Lean beef, well chopped and slightly roasted (inside rare), with potato purée, finely strained, the quantity not to be too small.

Afternoon. Like the morning diet, but without the egg.

Evening. Milk $\frac{1}{2}$ liter, or a plate of soup (as for breakfast), one roll with butter, and one or two soft-boiled eggs or scrambled eggs. A little wine is also permitted, also the addition of weak coffee or tea, bouillon, and chopped cold roast veal.

This is an absolutely bland and non-irritating diet, which as far as possible meets the requirements and the personal taste of the

patient, with no difficulties whatever in the way of procuring or preparation, as it includes only the simplest and always obtainable articles of food.

In spite of many objections this test diet has met with general approval. The objections were principally to the effect that the diet list was not the only possible one—which of course is correct. It is quite possible to compose a different test diet which would meet the demands laid down. But the value of the Schmidt formula lies in the fact that upon it as a basis, and through the labors of Schmidt, Strasburger, and their co-workers, our entire system of modern coprology and functional intestinal diagnosis has been constructed. All the numerous analyses and stool examinations which have furnished material for establishing systematic intestinal diagnosis have been made with this test diet. If at the present time we are able to speak of "normal feces," we are indebted for this achievement to the application of just this test diet. As soon, moreover, as we make quantitative or qualitative changes in the material points of this test diet, we rob the fecal examination of its firm and assured foundation and destroy the object of comparison—the normal feces—of which we must always avail ourselves in judging pathologic conditions.

As a rule, this test diet is well tolerated. Milk, possibly, might occasionally give rise to diarrhea. In such cases the milk is boiled together with cocoa or replaced entirely by the latter.

For purposes of examination the test diet is taken for two or three succeeding days, or at all events for a sufficient time to make sure of the fact that the stool is derived from it.

EXAMINATION OF THE TEST-DIET STOOL.

The test-diet stool is collected in a chamber, transferred to a glass or tin vessel specially reserved for this purpose, and sent to the physician. If the feces are hard or thick, a wooden spatula may be used for transferring them from the chamber; feces of fluid consistency may be poured into the receptacle for transportation.

Having thus obtained the test stool, the next step is its examination. This should be made macroscopically, microscopically, and chemically.

A. Macroscopic Examination.—The feces should always be examined as soon as possible after defecation. They should first be inspected and examined as to color, consistency, odor, and gross admixtures of mucus, blood, pus, and helminths.

The next step in the macroscopic examination is the trituration of the feces. (Plate VI.) This is done in the following manner: The entire quantity is thoroughly mixed, with a wooden spatula (Fig. 14, a), so that it becomes a homogeneous mass and it is certain that a sample taken from it represents the mixture. Of these

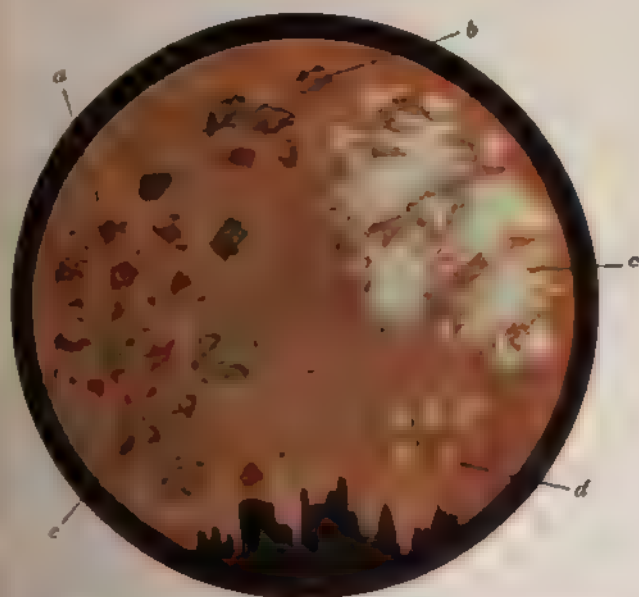
PLATE VI

FIG 1



Normal Test-diet Feces (Macroscopic).

FIG 2



Pathologic Test-diet Feces (Macroscopic)

a muscle, b potato, c connective tissue, d fat, e mucus

stirred feces a small portion, the size of a walnut, is carefully triturated in a mortar (Fig. 14, *b*), with gradual addition of water until the mass is of the consistency of soup. The trituration should be done so carefully that no coherent non-triturated particles will be visible to the eye. These feces, triturated to the finest possible consistency, are poured out and spread upon a black plate (Fig. 14, *d*), where it will be possible to observe with the greatest accuracy and distinctness whether any parts of the test diet, and which, have been evacuated in a macroscopically visible form—i. e., have not been digested and assimilated. The macroscopic examination, therefore, includes a search for connective tissue of the meat (Plate VI, Fig. 2, *c*), particles of muscle (Plate VI, Fig. 2, *a*), potato remnants (Plate VI, Fig. 2, *b*), fat (Plate VI, Fig. 2, *d*), and cellulose residue. Furthermore, in this examination it will be possible to recognize constituents which do not originate from the test diet, but from the intestine itself, as for instance the important matter of mucus (Plate VI, Fig. 2, *e*), small pus flakes, and large crystals of ammonio-magnesium phosphate.



FIG. 14.—Necessary apparatus for making analysis of feces: *a*, wooden spatula; *b*, mortar; *c*, watch crystals; *d*, black plate; *e*, fermentation tubes.

B. Microscopic Examination.—The microscopic examination serves to supplement the macroscopic, and requires three different procedures:

1. Inspection of a small particle of the untriturated feces, spread in a thin layer under the cover-glass: examination as to the presence of muscle particles, fat in its various forms, potato cells, cellulose remnants, cocoa remnants, mucus, pus, and parasite eggs.

2. A small particle of feces is thoroughly triturated on a slide with a few drops of a 30-per-cent. acetic acid solution, by means of a needle; heated for a moment over a flame to the boiling-point, and inspected under the cover-glass. By this process all the fat remnants are temporarily melted, the entire quantity of fat being shown in the warm preparation in the shape of liquid globules of fatty acid, spread over the entire surface. As the preparation cools, the drops coagulate into untransparent masses of fatty acid. From this preparation it is possible to approximately estimate the fat content of the feces.

3. A small particle of the feces is carefully triturated on the slide with a droplet of a strong compound solution of iodine (iodine 1, potassium iodid 2, distilled water 50), and inspected under a strong

light under the cover-glass. By this means any remnants of starch, either enveloped in cellulose or free, which stain blue with iodine, will be recognized. At the same time it is possible to observe any blue-stained iodine fungi and yellow-stained yeast cells.

C. Chemical Examination. 1. *Reaction Test.* The simplest method of testing the reaction consists in bringing a strip of red and a strip of blue litmus paper, soaked in water, into contact with the feces, and observing the change of color on the outer side of the paper. Schmidt recommends Azolitmin paper, which is prepared from the pure litmus coloring substance.

2. *Schmidt's Sublimate Test.*—The sublimate test serves to discover whether the feces contain the normal fecal pigment, hydrobilirubin, or pathologically unchanged biliary pigment (bilirubin). The test is based upon the fact that hydrobilirubin stains intensely tile-red owing to the formation of mercurial chlorid of hydrobilirubin, while bilirubin with sublimate stains green owing to the oxidation of the bilirubin, changing it to biliverdin. For this purpose it is necessary to triturate in a mortar (Fig. 14, *b*) a walnut-sized piece of feces to a thin consistency, adding a generous portion of concentrated aqueous sublimate solution (corrosive sublimate 25, sodium chlorid 25, distilled water 500), which is thoroughly mixed with the feces. The tile-red stain of hydrobilirubin will then rapidly occur with fresh feces (Plate VII, *a*). Feces which have been standing for some time produce a reddish-brown to a dirty brown color. The mixture should be allowed to stand for twenty-four hours, when any unchanged biliary pigment that may be present will be found to be stained green (Plate VII, *b*). In that case, either the entire quantity of feces is stained green or only a few macroscopic or microscopic green particles may be visible.

3. *Schmidt's Incubator Test.* The incubator test is instituted to show whether the feces incline to carbohydrate fermentation or to protein putrefaction, or whether they are negative. For this purpose Strasburger's fermentation tube (Fig. 15) is employed. This fermentation tube has a bottom vessel (*a*), into which 5 grams of formed feces are put with a wooden spatula and well stirred with water. If the stool is hard, a smaller quantity is taken, a larger one if it is liquid. The bottom vessel is closed with a perforated rubber cork, which is then connected with another and doubly perforated rubber cork carrying a small tube (*b*) filled with tap water, and connected by a piece of U-glass tubing with another small tube (*c*) which has an aperture at its upper end. The apparatus is kept in the incubator for twenty-four hours at a temperature of 37° C. Should gas develop, it will enter from the bottom vessel (*a*) into the tube *b*, displacing the water into the empty tube (*c*). Carbohydrate fermentation is assumed to exist if after twenty-four hours the outer tube (*c*) is about half-filled with water; if the reaction of the feces has become distinctly acid; if the feces

PLATE VII



Sublimate Test

a. normal feces b. pathological feces

in the bottom vessel, when it is opened, have an odor of butyric acid and their color has turned light yellow. Albuminous putrefaction has taken place if the reaction of the feces has become strongly alkaline, there is a distinct odor of putrefaction, the feces have assumed a dark color, and there is but slight development of gas. Baurmeister has modified the fermentation tube so that it is easily manipulated and more durable. The modification consists of three ordinary wide-mouthed bottles connected by glass tubing through perforated rubber stoppers (Fig. 13, *c*). The ground feces are placed in the first bottle, the second bottle is filled with water, and the third bottle remains empty. In the presence of fermentation or putrefaction, the generated gas forces the water with some of the feces into the third bottle. The degree of decomposition is determined by the quantity of fluid in the third bottle. When the test-diet feces are normal the second and third bottles remain the same as when first placed in the incubator (Plate VIII, *a*). In the presence of fermentation, some of the yellow feces are forced into the two other bottles (Plate VIII, *b*). The feces are usually darker in the presence of putrefaction (Plate VIII, *c*).

4. *Examination for Dissolved Protein.* For this purpose the following procedure is instituted. The feces (daily quantity) are well triturated, water being added slowly, and further diluted with water until a rather liquid consistency (about 500 Cc., or one pint) is obtained. This fluid is allowed to stand for a few hours and is then filtered through a double filter. The turbid filtrate is then passed for clarification through a silicated filter, which usually yields a clear filtrate. If it be desired to test the clear filtrate for dissolved protein

(peptone, albumose), it will be necessary to first remove the nucleoproteins which are present in every fecal extract. This is effected by the careful addition, in droplets, of a 30-per-cent. acetic acid solution to the liquid, in a test tube. The precipitated nucleoproteins cause a turbidity of the previously clear filtrate, which must now be passed through a double layer of filters. If the resulting filtrate is limpid and free from nucleoproteins, a few more drops of a 3- to 5-per-cent. solution of acetic acid should be added in order to make doubly sure that all of the nucleoproteins have been precipitated, after which the usual albumin test (boiling with acetic acid, the ring test with nitric acid, or the ferrocyanide-of-

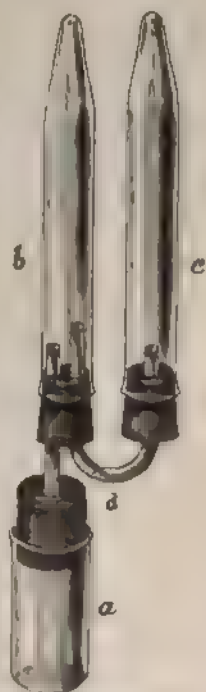


FIG. 13.—Straubinger's fermentation tubes.

potassium test) should be instituted. Should the filtrate, turbid from the precipitated nucleoproteins, remain so after the second filtration, it should once more be filtered through a silicated filter, which will clarify it and make it ready for examination for dissolved protein.

Quantitatively, the protein in the nucleoprotein-free fecal extract can be determined by Esbach's reagent, or Tsuchiya's 1-per-cent. solution of alcoholic phosphorous acid (Wolfram phosphorous acid 1, hydrochloric acid 5, 96-per-cent. alcohol 100). Tsuchiya has proposed special tubes, suitable for the Wolfram phosphorous acid test.

In this way every test-diet stool should be examined, particularly by the beginner. The entire examination as above described will not occupy more than a quarter of an hour at the most. As experience increases, the incubation test and the demonstration of dissolved protein may in many cases be entirely dispensed with, the macroscopic and if necessary the microscopic examinations allowing a sufficient survey of the functional condition of the intestine. The most important part of Schmidt's stool examination certainly is the macroscopic examination, which alone is usually sufficient for the experienced practitioner. Aside from Schmidt's technic of examination, the demonstration of blood and certain ferments in the feces is of importance. For these examinations a special test diet is not required.

Bacterial Preparations.—In order to separate the microorganisms from the other constituents of the feces, the following method has been evolved by Strasburger: A small quantity of feces, about the size of half a pea, is triturated with a few cubic centimeters of water and the mixture centrifugalized. The turbid liquid, containing bacteria, is poured off the sediment; one part is then diluted with two parts of 96-per-cent. alcohol and this mixture is centrifugalized. Of the resulting bacterial sediment a small quantity is placed upon the slide, the liquid is allowed to run off, and the bacteria are distributed in a uniform stratum upon the slide by covering the latter with a second slide, which is then drawn off. The result will be a very fine, even stratum, to be fixed over a flame. Staining agents are: Loeffler's methylene blue, a 10-per-cent. aqueous solution of carbolated fuchsin, Ziehl's carbolated fuchsin for the demonstration of tubercle bacilli, and a strong Lugol solution for staining granulose fungi.

THE NORMAL TEST-DIET STOOL.

It has already been pointed out how important it is for diagnostic purposes to know, and always to remember, the picture of the normal test-diet stool (Plate VI, Fig. 1) occurring in cases of intact gastric and intestinal function, since without such knowledge of the

PLATE VIII



Test-diet Feeds

a, normal; b, fermentation; c, putrefaction

also plays a rôle, because even in the normal chemism of the stomach connective tissue may appear in the feces when there is accelerated gastric motility. Even in hyperacidity there are occasionally remnants of connective tissue, and in these cases, provided the motility is normal or retarded, it will be necessary to think of insufficient secretion of pepsin, which may exist in spite of increased secretion of hydrochloric acid. It is the presence of connective tissue in the stool which in many cases renders a correct diagnosis possible, because even when intestinal catarrh and disturbed absorption coexist the connective-tissue remnants point to the stomach as the origin of these intestinal affections.

Muscle Remnants. If muscle remnants can be macroscopically detected in the feces, there is a digestive disturbance of the small intestine. The stomach is involved in only a minor degree in the digestion of meat. Muscle remnants can be recognized in finely triturated feces either as very small or as large red-brown lumps (Plate VI, Fig. 2, *a*). They can be smoothed out under pressure of the cover-glass and distributed with the needle. If there are macroscopic muscle remnants of this kind, the fact points to insufficiency of pancreatic secretion or absence of activating enterokinase in the secretion of the small intestine, or exaggerated peristalsis to such an extent that there is no time for digestion; or else a primary absorptive disturbance of the small intestine has led to decomposition and secondarily to diarrhea. It follows, therefore, that the appearance of muscle fragments alone is not sufficient to establish the disturbance of a definite intestinal function; all that can be said is that the disturbance must be looked for in the small intestine.

Macroscopic muscle remnants may under certain circumstances be absent while microscopically the feces will reveal exceedingly numerous and poorly digested muscle remnants. These findings have the same value as macroscopic muscle remnants, but should be weighed with even greater care.

If remnants of connective tissue and muscles are found in close proximity or even in contact with each other, the circumstance is proof of disturbed gastric and intestinal function.

Fat. Abnormal fat residues in the feces can be macroscopically recognized from their glistening, clay-like appearance (Plate VI, Fig. 2, *d*). Again, when the feces are finely triturated with water, there is frequently a fatty membrane visible at the surface. The presence of much fat imparts to the feces the well-known clay color. In the microscopic acetic acid preparation the presence of increased fat is assumed if the droplets of fatty acid in the field are materially increased in comparison with those of normal feces.

Pathologically, fat occurs in the shape of fatty acid needles and neutral fat. The fatty acid needles are long, thin, finely curved, and oftentimes arranged in rays. Soap needles, on the other hand,

are smaller, thicker, and more compact. Neutral fat occurs in the shape of light-colored or yellowish drops and scales.

The occurrence of fatty stools is commonly associated with insufficiency of biliary secretion or impeded biliary flow into the intestine (icterus). In disturbed pancreatic secretion, pronounced fatty stools likewise occur; in this case the fat is present mostly as neutral fat. Furthermore, fatty stools will occur in disturbed intestinal digestion, especially in grave organic affections such as tuberculosis, amyloid degeneration, or typhus mesenterica.

Carbohydrates. Potato Remnants.—These are macroscopically visible in finely triturated feces as isolated and usually very numerous potato cells or as small, sago-like, coherent potato cells (Plate VI, Fig. 2, *b*) which can microscopically be recognized as potato from their size and shape (large, roundish). From the microscopic examination the condition is considered pathologic if there are numerous potato cells in the field of vision. The pathologic potato remnants usually contain starch, which is microscopically demonstrable in the iodine preparation.

Starch.—If there is a disturbance in the carbohydrate digestion, free starch granules are usually to be found, either whole or fragmented, and visible in the iodine preparation.

Insufficient starch digestion is chargeable to the small intestine, and is probably caused by a disturbance of the secretory function of that section of the intestinal tract.

B. Pathologic Products of the Intestinal Wall.—*Mucus.*—The presence of mucus in the feces is always a sign of an inflammatory condition of the intestinal mucosa, with the exception of that viscous brownish mucus which often adheres to the feces and originates in the rectum, and of the mucus that is often profusely evacuated in enteritis membranacea. As a rule the mucus can be made plainly visible in the finely water-triturated feces poured out on a black plate; it appears in large or small flakes which are transparent upon the black ground, capable of being moved to and fro with the needle, and separated only with difficulty (Plate VI, Fig. 2, *c*). Oftentimes, however, it is not an easy matter to determine whether the mucus originates in the small or in the large intestine. Generally speaking, mucus from the large intestine is large-flaked, often of a whitish turbidity, and usually contains numerous well-preserved intestinal epithelial cells. Mucus originating from the small intestine, on the other hand, contains no epithelia, but at the most a few undigested cell nuclei and very many bacteria. If the mucus has a labary tint or stains green under the sublimate test, the probability is that it is from the small intestine. Microscopically it is often possible to find minute bilirubin crystals in the small mucous flakes from the small intestine.

Soluble Protein.—The small mucous flakes of the small intestine are often dissolved before they leave the intestine with the feces,

so that a search for them in the feces is fruitless. In that case the chemical examination of the feces for protein offers an equivalent substitute. Very frequently a pronounced decomposition of protein (incubator test), or dissolved protein, is found in cases of intestinal inflammation (Plate VIII, c). This finding is by no means a result of decomposition of undigested remnants of food, but indicates a putrefaction of the pathologic albuminous products of elimination on the part of the inflamed mucosa itself. A product of this kind is mucous, but more particularly serous, thrown into the intestinal lumen by transudation in inflammatory conditions. Thus, the demonstration of the putrefactive character of a stool is equivalent to a demonstration of mucus. Mucus, putrefaction, and the presence of soluble protein are the characteristics of the stool in catarrh of the small intestine; and on the ground of these findings we are often in a position to make a diagnosis of catarrh of the small intestine at a time when there is nothing but a slight inflammation, and no pronounced symptoms are apparent.

Suppuration.—Suppuration occurs in grave catarrh and especially in ulceration of the intestinal canal. It is visible in the finely triturated stool on a black ground in the shape of small, lentil-shaped, yellowish-gray flakes, which microscopically turn out to consist of purulent matter.

C. Unchanged Biliary Pigment (Bilirubin).—It is a pathologic sign if, with the sublimate test, the entire quantity of feces under examination or only a microscopic part of it stains green (Plate VII, b). It is proof that the bilirubin, after having entered the cecum, has not been normally reduced to hydrobilirubin. This defect may occur when the motility of the intestine is not particularly exaggerated or when, for some reason, the normal process of reduction is absent. As a rule the green coloration of the feces is a sign of pathologic involvement of the small intestine.

If a fecal specimen, when subjected to the sublimate test, does not stain at all, the fact proves the total exclusion of bile from the intestine.

D. Bacteria.—The presence of tubercle bacilli may be demonstrated by the described method of Strashburger for isolating bacteria from the feces. Tubercle bacilli have a predilection for mucous flakes and purulent matter if such occur in the stools. The granulose iodine fungi, as Schmidt termed them, which are nearly always present, may be observed in the microscopic iodine preparation in cases of questionable carbohydrate fermentation of the feces. Furthermore, the iodine preparation admits of the easy recognition of yeast, lactic acid bacteria, and sarcinae, which stain yellow with iodine.

THE DEMONSTRATION OF BLOOD IN THE FECES.

Admixtures of large quantities of blood are macroscopically recognizable by the well-known tar-black color of the feces.

The following chemical methods serve to demonstrate the smallest occult hemorrhages, such as are of frequent occurrence in gastric and duodenal ulcer, and the diagnosis of which is sometimes decided by them. Blood originating from the stomach or the upper intestinal tract is excreted as hematin; on this fact is based the chemical demonstration of blood.

Hematin can be found with the spectroscope. Ether, which in the presence of hematin is of a brownish hue, will show the spectrum of hematin. This has an intense, narrow stripe of red between C and D, but considerably less pronounced than the latter; also three stripes of yellow on the borderlines between yellow and green and between green and blue. The latter, as a rule, can only with difficulty be distinguished.

Occult Blood.—It has been known for some time that in ulcer or carcinoma of the stomach there is always slight gastric hemorrhage. Clinicians have been unable to tell definitely whether the blood found in the stomach contents came from the neoplasm or was due to irritation by the stomach tube. The slightest irritation by the stomach tube would produce a small amount of invisible blood, which would respond to the chemical test. Bous was the first to examine the feces for invisible blood; he knew the blood corpuscles would degenerate, and the hematin crystals should be found in the feces. If these hematin crystals could be found in the feces, with other signs of either ulcer or carcinoma, the test would be valuable. Examination for invisible blood in the feces is of great importance, since many clinicians report the constant presence of occult blood in cases of gastric ulcer and carcinoma. The term "occult blood" is applied to minute hemorrhages discharging in the gastro-intestinal canal, too small to be discerned macroscopically. By the time the blood passes through the whole intestinal canal the corpuscles are so broken down that they cannot be found with the microscope. The tests for occult blood are chemical since they are extremely sensitive, very small amounts of blood can be detected. There is great liability to error in determining the site of the hemorrhage. Besides epistaxis, hemoptysis, and hemorrhoids, any foodstuff containing hemoglobin in any amount will give the reaction. Therefore the diet must be a blood-free one for three days previous to the test.

Benzidin Test for Occult Blood.—This test is very sensitive. It was first described by O. and R. Adler, and has been subjected to slight modifications by Schlesinger and Holst. The test is made as follows:

1. One gram of benzidin is placed in a test tube with about 2 Cc. of glacial acetic acid. This mixture should be freshly prepared.

II. A small piece of feces (the size of a pea) is rubbed up with water and placed in a test tube and boiled; boiling destroys the oxidizing ferments.

III. To about 3 Cc. of peroxid of hydrogen in a test tube, add about five drops of the above glacial acetic acid-benzidin mixture, and lastly a few drops of the boiled feces.

Blood is indicated by a greenish or blue color (Plate IX).

Phenolphthalein Ring Test for Occult Blood. Boas prefers the phenolphthalein test for occult hemorrhages of the gastro-intestinal canal. He points out that the benzidin test, which demonstrates blood in a dilution of 1 to 200,000, is altogether too delicate; it indicates the minutest quantities of alimentary (exogenous) blood, and the reaction may be distinctly positive after three or four days of meat-free diet. Weber's guaiac test (page 86) is less susceptible, but, inasmuch as the substances which disturb the test must be extracted by alcohol and ether, there are certain difficulties in the way of its practical application not present in the phenolphthalein test. The phenolphthalein test is based on the fact that phenolphthalein, in an alkaline solution, is oxidized by blood pigment so that the solution becomes pink or red. The phenolphthalein reagent is prepared as follows: 1 gram of commercial phenolphthalein and 25 grams of potassium hydrate are dissolved in 100 Cc. of water, and to this solution 10 grams of zinc powder are then added. The mixture, which is at first red, is boiled over a small flame (about two hours if necessary) under constant stirring and shaking until complete decoloration has taken place by reduction. The hot solution is then filtered. For preservation a small excess of zinc powder may be added.

The demonstration of blood is carried out as follows: Fifteen drops of phenolphthalein reagent are put into a beaker and five or six drops of a 3-per-cent. hydrogen peroxid solution added. Then add 2 Cc. of absolute alcohol, and shake. Some of the extract of the feces (made by extracting them with a mixture consisting of five drops of glacial acetic acid and 15 to 20 Cc. of absolute alcohol) is then filtered through a common filter into the reagent glass, the funnel being so placed with respect to the glass that the filtrate will come in contact with the solution at the margin. If blood be present, a pink or deep red ring will appear at once or gradually, according to the amount of blood. By placing the beaker on a pure white surface, the red line is brought out more distinctly by contrast. The principal advantage of this test consists in the characteristic reaction, the possibility of differentiating between the presence of large and small quantities of blood, the permanency of the reagent, and simplicity of preparation.

In point of acuity, the phenolphthalein test stands between Weber's guaiac and the benzidin test. If the benzidin or phenolphthalein test is to be employed, a preparatory period of several days of meat-free diet must be insisted upon.

PLATE IX



Fig. 1. Fig. 2.

Scale of feet.

THE DEMONSTRATION OF FERMENTS IN THE FECES.

In intestinal affections in which the differential diagnosis of a pancreatic affection is involved, it is important to examine the stool for pancreatic ferments, as their absence or presence will decide the diagnosis under certain circumstances.

Trypsin.—*The Plate Test of Müller-Schlecht.*—If a few small drops of the test material be placed upon the surface of a so-called Lœffler serum plate, and this plate be kept in an incubator at a temperature of 50° to 60° C., there will occur slight but gradually increasing indentations if trypsin be present. In the absence of the ferment juice there will be no such indentations.

The Casein Method of Gross.—The principle of this method is based upon the fact that casein, which is easily soluble in a weak alkaline solution, is readily precipitated upon the addition of dilute acetic acid. For purposes of fecal examination a 0.5-per-cent. solution of casein is prepared by dissolving 0.5 Gm. of pure casein (Gruebler) in one liter of a 1-per-cent. sodium hydrate solution by heat. The feces are mixed in a mortar with a treble quantity of a 1-per-cent. sodium hydrate solution until a uniform mass is obtained, and filtered until a clear yellow filtrate appears, which is usually in a short time. If the resulting filtrate is not quite clear, the turbid element is allowed to settle and the supernatant clear liquid is used. One hundred cubic centimeters of the casein solution is placed in a small retort, 10 Cc. of the fecal preparation added, and the mixture placed in a thermostat at 38° to 40° C. By taking samples from time to time it will be found that no more turbidity occurs upon the addition of a 1-per-cent. acetic acid solution when all of the casein has been digested—proving the presence of trypsin.

It has been found that in all cases where there is no affection of the pancreas or occlusion of the pancreatic excretory ducts, trypsin is demonstrable in the feces. In order to obtain a high percentage of trypsin in the feces, a strongly protein diet or a mild laxative should be administered. The digestive period of casein is between eight and fifteen hours; usually it is from twelve to fourteen hours. It is possible to draw approximate quantitative conclusions from appropriate fecal dilutions.

Steapsin.—To 10 Cc. of distilled water in a small flask are added 1 Cc. stool filtrate, 1 Cc. ethyl butyrate, and 1 Cc. toluol. One drop of a 1-per-cent. alcoholic solution of phenolphthalein is now added, and the mixture made neutral with a decinormal sodium hydrate solution. Add water to bring the fluid to 25 Cc., cork, shake, and incubate for twenty-four hours at 40° C. After incubation the mixture is titrated to neutral again with a decinormal sodium hydrate solution. A control is made by using stool filtrate which has been boiled for five minutes. The difference between the amount of free acid which has developed in the control

flask and that which has developed in the test flask is an index of the fat-splitting ferment present in the latter.

Demonstration of the presence of steapsin in the intestinal contents is also made by either of two methods, the Grutzner-Gamgee or the von Oefele.

By means of the former the fat-splitting ferment manifests itself by the change in color which it produces in a mixture of oil-gum emulsion and neutral litmus solution. The emulsion consists of 10 parts of oil, 5 of gum, and 35 of water; the litmus solution in 12-mm. test tubes shows violet against white paper. In each of several such test tubes 10 Cc. of the litmus solution and 5 drops of the emulsion are placed, and graded amounts (from 2 to 32 drops) of the fluid to be tested are added. The tubes are then placed in a water-bath at 37° C., and after a few minutes examined. Redness, more or less pronounced according to the amount of intestinal fluid added, is proof of the presence of the fat-splitting ferment.

By von Oefele's method the steapsin in the intestinal juice tested dissipates the red tint of a mixture of melted sweet butter, potassium carbonate and phenolphthalein titrated with a soda solution until it becomes red. The melted butter is mixed with an equal amount of a 1-per-cent. aqueous solution of potassium carbonate, with a little phenolphthalein added. Heated to 55° C., 5 Cc. of this liquid is then shaken in a warm test tube with 5 drops of intestinal fluid. Normally the red color will disappear in two to five minutes, as a result of the action of the steapsin present.

The Nuclei Test of Adolf Schmidt.—Schmidt found that the cellular nuclei, unlike the connective tissue, are digested by the pancreatic secretion only, and not by the gastric juice. If, therefore, undigested tissue nuclei reappear in the feces, we may conclude that the pancreatic function is defective.

The test is made in the following way: The patient swallows for several consecutive days a small cube of meat enveloped in a bag of silk gauze. These silk bags are easily recognized in the feces, especially if the tying silk thread be long. The meat remnants contained in the little bags are examined for the presence of nuclei, either in their original condition by treatment with acetic acid or methylene blue, or, after hardening, in stained sections.

The meat cubes are cut from fresh meat; they are about one-half centimeter long and are preserved in alcohol. After hardening, they are placed in the small silk bags and again preserved in alcohol. Before use, the bags, with their contents, should be dehydrated for several hours. Strauch¹ has verified the nuclei test in Abderhalden's Physical Institute with pure digestive juice obtained from a dog through a stomach fistula, and arrived at the following results: Pure gastric juice (pepsin and hydrochloric acid) leaves

¹ Deutsches Archiv f. klin. Medizin, vol. cv, Nos. 1 and 2.

tissue nuclei unchanged. Pure pancreatic juice (trypsin) completely dissolves cell nuclei within six to eight hours. Pure intestinal juice (erepsin) does not influence cell nuclei. Juice pressed from the intestine will dissolve cell nuclei, but only at a slow rate. Thus the correctness of the basis upon which Schmidt's nuclei test rests has been demonstrated.

Diastase Test (Wohlgemuth).—The diastase is demonstrated by testing the dextrinizing influence of a present diastase upon a starch solution, an iodine solution serving as indicator.

This is done, according to Wohlgemuth, in the following manner: A weighed quantity of 5 grams of fresh feces is triturated in a mortar with 20 Cc. of a 1-per-cent. solution of sodium chlorid; at first only a few cubic centimeters of the measured quantity of sodium chlorid solution are added to the mass, which is triturated until a perfectly homogeneous product results; and this is continued until all of the liquid has been triturated with the feces. This mixture is allowed to stand at room temperature for thirty minutes, during which time it should be frequently stirred up. The liquid mass is then uniformly distributed into two centrifuge tubes (10 Cc. in each) exactly marked and graduated. The tubes are rotated until the solid parts have settled, which will be in five to ten minutes, and the height of the solid sediment and of the liquid is read off the graduated tubes and noted. The supernatant ferment-containing fecal extract is poured off, and the diastase determined by a series of tests. For this purpose the liquid is distributed over nine test tubes: the first three tubes will receive 1, 0.5, and 0.25 Cc., respectively, of the undiluted fecal extract, and this procedure is continued so that each succeeding tube will receive one-half the quantity contained in the preceding one. This object is most conveniently attained if an eighth and a sixty-fourth dilution of the original fecal extract be worked with. Tubes 4, 5 and 6 will then receive 1, 0.5, and 0.25 Cc. of the one-eighth dilution; tubes 7, 8 and 9, 1, 0.5, and 0.25 Cc. of the one-sixty-fourth dilution. All the dilutions are made with a 1-per-cent. solution of sodium chlorid, and all the tubes are brought up to 1 Cc. in volume by the same solution in order to obtain throughout a uniform concentration of sodium chlorid. Then 5 Cc. of a 1-per-cent. starch solution is added to each test tube. The tubes are then tightly closed with a cork or cotton plug and kept in the incubator at 38° C. for twenty-four hours. This period having elapsed, they are taken out and cold water from the tap is added up to a finger's width from the bottom. To each, one drop of a decinormal iodine solution is added, and the lowest limit of diastatic efficiency is exhibited by the tube in which a blue coloration appears first.

Indigestion Tests.—Steatorrhea, or increased fat in the stools, occurs not only in icterus, but in pancreatic disease. The stools are of a light color, often voluminous and of a rancid odor; if many

fat crystals are present they may impart an aluminum-like appearance to the stools. A phenomenon that is said to be quite characteristic of defective pancreatic secretion is the passage of colorless liquid stools which harden on cooling. The bile which normally finds its way into the intestine prepares the fats, by emulsification, for the action of the pancreatic lipase; so that even though as much as 60 per cent. of the fats ingested be found in the stools, there is not necessarily any pancreatic defect—the fault may be with the liver. Normally, as much as 10 or 11 per cent. of the ingested fats passes through the intestine unchanged; an amount in excess of this indicates icterus or pancreatic insufficiency. To estimate the amount of fat in the stools, both microscopic and chemical methods are employed—the latter in case the former is inadequate. The microscope reveals the fat as droplets, needle-like crystals, or flakes. For accurate quantitative estimation of fats, the feces are dried on a water-bath, the fatty acids and neutral fats extracted with ether, the residue treated with dilute hydrochloric acid to convert any soaps that may be present into fatty acids, and these in turn extracted with ether. By measuring the dried feces to be treated, percentage results are obtained.

The fat-splitting function of the pancreas may be tested with artificial compounds of iodine and fat, for steapsin possesses the power to decompose such compounds. If 5 Cc. of calcium mono-iodobehenate (sajodin) be taken with the meal, iodine will, we are told by Syring, appear in the urine within three to five hours in all normal individuals. Icterus as well as pancreatic insufficiency, however, prevents its appearance. The iodine test was introduced by Wintermütze.

Carmin or Charcoal Test.—In order to determine the motility of the intestine, certain substances which color the feces are given at a definite time, and the evacuations are watched to ascertain the interval necessary for these substances to appear. Charcoal colors the feces black. Carmin colors them red. By administering a dose of charcoal or carmin at the commencement of an intestinal test diet, we are able to mark off the moment when it begins to appear in the stools, and when we may safely use the latter for purposes of examination. We may mark off and obtain the total feces corresponding to a test diet extending over a fixed period by giving a carmin capsule 0.5 Gm. ($7\frac{1}{2}$ grains) at its commencement and another at its termination. As Basch remarks, in the carmin test we have a simple, harmless, reliable and convenient means for the demarcation of stools and the estimation of gastro-intestinal motility and patency, for the detection of fistulous communications of the alimentary canal with the exterior or with other hollow organs, for the location of the distal end of a duodenal tube, and to aid in the differentiation between esophageal diverticulum and dilatation.

following conclusions may be drawn: In case all the beads emerge in a much shorter time than twenty-four hours, there is an accelerated motility; if they emerge after forty-eight hours, a retarded motility exists. The digestive function is good if all the beads are empty or if there are but traces of fat or thymus (also fishbone) left. A reappearance of catgut or meat, potato, much fat or much thymus, always indicates a poor digestive function for the food substance in question. If all these test substances reappear in the stool, an absolutely poor digestive function exists.

Preparation of Food Beads.—(1) *Catgut*: Take raw catgut No. 00, draw it through the bead, and tie the ends together (Plate X, a). (2) *Fishbone*: As the ordinary fishbone breaks when tied in a knot, it is best to use the long bones from a pickled herring. The bones are washed in water first, then rubbed off with a cloth, and kept in water in a bottle. When wanted, they are taken out of the water, drawn through the bead, and tied in the same manner as the catgut (Plate X, c). (3) *Meat*: The muscle fibers of raw beef are cut lengthwise in the direction of the fibers and in pieces 5 to 6 centimeters (about 2 inches) long, and 1 centimeter ($\frac{1}{2}$ inch) thick. These are preserved in a bottle of alcohol. Take a piece of meat from the alcohol bottle, tear off lengthwise a muscle fiber 2 or 3 centimeters long and 1 millimeter ($\frac{1}{16}$ inch) thick, draw it through the bead and allow the ends to overlap; next tie the ends fast together over the bead with a silk thread (Plate X, e). (4) *Thymus*: Raw sweetbread from the calf is cut in cubes and preserved in alcohol. For use, lay a small piece, about 2 cubic millimeters, within a small square of gauze, fold the four ends of the gauze together and tie with thread, so that the small piece of thymus lies enclosed as in a purse; then fasten the gauze purse to a bead (Plate X, b). (5) *Mutton fat*: Beads with a large opening (1.5 or 2 millimeters in diameter) should be dropped in hot rendered mutton fat and after a minute taken out with a forceps and placed in a vessel of cold water. This congeals the fat. Then they are laid on a piece of pure filter paper and allowed to remain until thoroughly dried. The beads can then be kept as long as desired, and are ready when wanted for use (Plate X, f). (6) *Potato*: Cook a piece of potato with the peel on in boiling water two minutes. Take out of the water and cool. Now cut a small piece of the potato with peel, 1 centimeter ($\frac{1}{2}$ inch) long, 0.5 centimeter wide, and 1.5 or 2 millimeters ($\frac{1}{8}$ or $\frac{1}{4}$ inch) thick, and attach it to a bead (Plate X, d). Two or more food substances may be attached to one bead; for instance, catgut and fishbone, meat and thymus. The test beads can all be kept on hand with the exception of the potato, which must always be freshly prepared. Meat and thymus beads are best kept in alcohol. Catgut, fishbone and fat beads are simply preserved dry. The beads, when prepared, should be strung together, and the string placed in a gelatin capsule and

PLATE X



Bead String.

a, catgut bead, *b*, thymus bead, *c*, fishbone bead; *d*, potato bead; *e*, meat bead; *f*, mutton-fat bead, *g*, bead-test capsule.

so administered, best shortly after a meal (Plate X, *g*). The bead test gives a very fair idea of how long the food remains in the intestinal tract, which the test diet does not. The bead test is designed to show the digestibility of protein, fat, and carbohydrate, and the motility of the gastro-intestinal tract.

THE TEST-DIET STOOL FINDINGS IN GASTRIC AND INTESTINAL AFFECTIONS.

ACHYLIA GASTRICA AND SUBACIDITY.

(a) *Slight gastrogenic disturbances of the intestine, or none at all.* The exterior aspect of the stool resembles the normal in all respects. Connective tissue is more or less abundant. On microscopic examination small connective-tissue fibers are seen, which may be the expression of insignificant absorptive disturbances of the small intestine; fat somewhat increased, fatty soap needles, increased muscle fibers. Chemical examination normal.

(b) *Pronounced gastrogenic intestinal disturbances.* In this condition the stool loses its concentrated form and is thin or diarrheic; for this reason a uniform picture of the stool cannot be given. Macroscopically there is always some connective tissue, which frequently coheres with the meat remnants. At the same time the greatest variety of catarrhal conditions of large and small intestine, fermentation of carbohydrates, or protein putrefaction, may exist. The demonstration—if need be, microscopic—of yeast, sarcinae, and long bacilli, is of importance.

HYPERACIDITY.

Feces either normal, or pathologic as in atonic constipation. In rare cases connective tissue (pepsin insufficiency?)

GASTRIC ULCER AND GASTRIC CARCINOMA.

Occult blood.

CHRONIC CATARRH OF THE SMALL INTESTINE.

(a) *Mild Cases.*—Stool formed or massy. Small potato remnants, isolated potato cells. Minute muscle fragments. Fat increased. Small mucous flakes of the small intestine.

Microscopic: Abundant, poorly digested muscle fibers. Abundant potato cells, with and without starch. Fatty soap and fatty acid needles.

Chemical: Reaction increased alkaline or acid, according to the predominance of protein putrefaction or carbohydrate fermentation.

Sublimate test: Red coloration.

Incubation test: Negative, or protein putrefaction. Rarely carbohydrate fermentation.

Dissolved protein: Negative, or weakly positive.

(b) *Grave Cases*.—Thin, watery stool, of green color in the more serious conditions, otherwise dark brown, malodorous. Meat remnants small or large. Numerous isolated potato cells and rather large potato pieces. Much fat, possibly fat lumps. Abundant mucous flakes of the small intestine, which may show a biliary discoloration.

Microscopic: Abundant muscle fibers, oftentimes strongly saturated with bile. Potato cells with and without starch. Free starch granules. Abundant fat (fatty acid, soap needles, also neutral fat in some of the severest cases). Minute mucous flakes. Ammonio-magnesium phosphate (protein putrefaction).

Chemical: Reaction alkaline.

Sublimate test: Green coloration either of the entire feces or of some macroscopic or microscopic particles.

Incubation test: Pronounced putrefaction. Rarely fermentation.

Dissolved protein: Strongly positive.

Bacteria: Cocci in protein putrefaction, proteus, *Bacillus fluorescens*, etc.

CHRONIC CATARRH OF THE LARGE INTESTINE.

(a) *Mild Cases*. Stool formed, massy, or thin, of normal color. Large and medium-sized mucous flakes of the large intestine.

Microscopic: Nothing of consequence.

Chemical: Reaction normal.

Sublimate test: Red coloration.

Incubation test: Normal, or slight decomposition.

Dissolved protein: Perhaps weakly positive.

(b) *Grave Cases*.—Diarrheic, watery stools, malodorous, of dark brown or more frequently light color. Large quantities of mucus in large and small flakes, non-transparent, whitish, sometimes sanguineous.

Microscopic: No peculiarities to notice.

Chemical: Reaction usually alkaline.

Sublimate test: Red coloration.

Incubation test: Putrefaction.

Dissolved protein: Positive.

Frequently both the small and the large intestine are inflamed, and the feces are correspondingly composed; they may be construed from the above stool pictures. The feces in acute catarrh of the small and the large intestine also correspond to the description given above. If achylia or subacidity is present, the findings of connective tissue should be added in order to obtain the stool picture of gastrogenic diarrhea.

DYSENTERY.

In dysentery there is the picture of the gravest kind of catarrh of the large intestine complicated by abundant admixture of blood in the stool, and sanguineous mucus (red dysentery) or pus (white dysentery). Oftentimes the intestinal evacuation consists of nothing but pus, blood, and mucus, mixed with small particles of feces. If there is simultaneous catarrh of the small intestine, the symptoms of disturbed digestion will be present in addition.

Chemical: Reaction alkaline.

Incubation test: There is, of course, considerable protein putrefaction.

Bacteria: In endemic dysentery the *Endameba histolytica*, *Endameba tetragena* or the *Endameba coli* of Loesch is present and can be seen in motion in the mucous flakes under the warm microscope. Occasionally the *Balantidium coli* is found. In epidemic dysentery there is the *Bacillus Shiga-Kruse-Flexner*.

INTESTINAL TUBERCULOSIS.

The test-diet stool presents the picture of grave catarrh of both the large and the small intestine. Further findings are:

Macroscopic: Pus.

Microscopic: Pus.

Chemical: The blood test is often positive.

Bacteria: Tubercle bacilli in the bacterial sediment, mucus and pus flakes.

DUODENAL ULCER.

Normal feces. Constipation stool. Occult blood.

ENTERITIS MEMBRANACEA.

The characteristic sign consists in the evacuation of mucus in large flakes and strings, sometimes without any feces. However, the feces may resemble completely the normal stool, although in most cases there is constipation stool (see Atonic and Spastic Constipation). Less frequent are thin and diarrhetic stools with no signs of disturbed digestion of the small intestine.

ATONIC CONSTIPATION.

A very small quantity of hard, dark brown, odorless scybala. Over their surface there is occasionally a brownish, lac-like film, with small traces of fresh blood, emanating from hemorrhoids.

Macroscopic: No food remnants, nor cellulose remnants such as occur in the normal feces.

Microscopic: As compared with the normal feces, there are surprisingly few remnants of muscle fiber, fat, or potato cells; and there are also fewer bacteria.

Chemical: Reaction neutral.
Sublimate test: Red coloration.
Incubation test: No reaction.
Dissolved protein: Negative.

SPASTIC CONSTIPATION.

In spastic constipation the feces usually correspond to the normal feces; they are evacuated in the shape of small or large hard balls and cylindrical pieces (sheep feces), which are often covered with a mucous film.

INTESTINAL FERMENTATION DYSPEPSIA.

The stool is either thin or massy, of yellow-green or golden-yellow color, of an intensely acid odor, and permeated with gas bubbles. Large quantities of potato cells are found, either isolated or coherent in the shape of sago clumps.

Microscopic: Numerous potato cells in the field of vision, filled with starch, in the iodine preparation. Numerous free starch granules.

Chemical: Reaction strongly acid.
Sublimate test: Red coloration.
Incubation test: Strong fermentation.
Dissolved protein: Negative.

Bacteria: In the iodine preparation numerous iodine fungi, stained blue. Here again there may be combinations with conditions of catarrh or achylia, which would effect a corresponding change in the stool picture.

NERVOUS DIARRHEA.

The stool either consists of thin masses or is of fluid consistency, having the appearance of fresh contents of the small intestine.

Macroscopic examination reveals numerous undigested food remnants (muscle, potato, fat, cellulose).

Microscopic: Numerous undigested food remnants of all kinds.

Chemical: Reaction normal.

Sublimate test: Red coloration or, under certain circumstances and considerably accelerated evacuation, green coloration.

Incubation test: Negative.
Dissolved protein: Negative.

STENOSES AND INTESTINAL CARCINOMA.

The stool pictures vary considerably according to whether there is diarrhea or chronic constipation, which conditions may again be complicated by catarrh. Of importance are admixtures of blood, sanguineous mucus and pus, which may also be macroscopically visible. Furthermore, occult blood is to be considered.

CHAPTER V.

ROENTGEN-RAY EXAMINATION.

By filling the stomach and intestine with a substance opaque to the Roentgen ray, we can determine the size, shape, position and motility of the stomach, and visualize the outline of the intestinal tract. The patient partakes of a meal of about 300 grams, containing about 60 grams of bismuth subcarbonate or barium sulphate. For holding these salts in suspension, buttermilk is usually employed, although potato-flour soup or thin porridge can be used. For the detection of perforating ulcer of the stomach and to permit of a more accurate bringing into relief of small defects, it is now considered advisable to use a plain water suspension of barium for at least the first half of the meal. For an accurate roentgenographic examination of the stomach, the patient should be prepared by fasting, so that at the time the bismuth or barium meal is given the stomach is entirely empty. The examination should not be made while the patient is under the influence of a laxative drug of any kind, since laxatives interfere with the ordinary functioning of the gastrointestinal tract. By making the first examination in the morning the behavior of the stomach can be observed during the period of emptying. If time is an important consideration, as in hospital observations where a number of cases have to be examined, the so-called double meal can be employed; the patient is given the first meal very early in the morning, and examined after five or six hours to determine the time of emptying; then the second meal can be given and the motility of the stomach observed. Usually, inspection with the fluoroscope will consume several minutes, the ray being turned off and on a number of times. The second examination is usually made at the end of two hours after the second meal and again three hours later. Observations are also made at the end of twelve and twenty-four hours respectively and at such intervals as will enable the roentgenologist to obtain a comprehensive idea of the course of the meal from its ingestion to its complete expulsion.

For the demonstration of small lesions about the pylorus and for the proper visualization of the duodenal bulb if it shows spasm or defects, it is essential that manipulation of the stomach be practiced during fluoroscopy. This can be done either with a wooden spoon or preferably with the hands, protected by the ray-proof glove. The manipulation of the stomach serves to accentuate defects and to overcome spasm. If an organic lesion is present the

stomach will at once resume its pathologic contour after manipulation has been practiced.

Roentgen fluoroscopy supplements the roentgenogram, as it gives immediate information regarding the changes in position and movements of the stomach and intestine; the observer sees these changes while they are being enacted. For the detailed study of minute pathologic alterations, such as small lesions about the pylorus and duodenal cap, the roentgenogram is more precise; the record is permanent. By taking serial roentgenograms at short intervals we obtain most valuable information. By comparing the erect with the Trendelenburg position, the presence of adhesions and the effect of ptosis upon intra-intestinal movement may be quite accurately determined.

The technic of the application of the Roentgen ray to the diagnosis of diseases of the digestive tract is similar to that employed in examination of the body for surgical Roentgen pathology. It cannot be taught in books, but needs months of study in well-appointed Roentgen laboratories.

Roentgenography of the stomach, esophagus, and intestine both large and small, shows the form of these organs, their deviation from normal with respect to size and contour; furthermore, their position and action as governed by muscular contractions, so that either the normal physiology may be established or the pathologic diagnosis may be arrived at. Oftentimes the dividing line between the physiologic and the pathologic appearance is slight; nevertheless the Roentgen-ray examination of the gastro-intestinal tract furnishes definite information which cannot be acquired with as much certainty by any other method. While few organic lesions present an absolutely pathognomonic appearance when viewed singly, yet a complete examination of the gastro-intestinal tract by the fluoroscopic method, assisted by manipulation and combined with the serial plate procedure, will rarely miss important pathology.

Spasm in different portions of the gastro-intestinal tract, especially with regard to the pylorus and the greater curvature of the stomach, is often mistaken for an organic lesion. Atropin relieves spasm and should always be employed as a routine measure in every case where spasm is suspected; it can be given hypodermically in doses of 0.0006 Gm. ($\frac{1}{100}$ grain).

EXAMINATION OF THE ESOPHAGUS.

While the application of the Roentgen ray in the diagnosis of diseases of the alimentary tract was first practiced by F. H. Williams, of Boston, Holzknecht demonstrated in particular that an oblique transillumination of the esophagus (from the right posteriorly to the left anteriorly, or from the left posteriorly to the right anteriorly) permits convenient observation of the entire esophageal

tract. The condition of the lumen of the gullet can thus be closely studied during rest, during respiration, and during deglutition. The pharynx and the upper portion of the esophagus may also be exposed to the Roentgen ray, illuminating the throat in a transverse or oblique direction.

Roentgen-ray examination of the esophagus gives most accurate and constant findings. In order to obtain a constant shadow of the esophagus, there must be an obstruction. Recently the use of a lumen-filled "hog casing" has given early information of slight indentations and stricture. Bismuth suspended in syrup of acacia, making a very thick emulsion, gives perhaps the most complete outline of the esophagus, inasmuch as the syrupy mixture tends to adhere to the esophageal walls. The details of this method have been perfected by Hirsch, of New York. Besides the presence of intrathoracic masses, a roentgenogram will show obstruction due to changes in the walls of the esophagus, as stricture, benign and malignant growths, aneurysm, diverticulum and spasm. The esophagospasm may be due to ulcer, to carcinoma, or to a neurosis, as in cardiospasm.

Spasm of the Esophagus.—A spastic stricture does not present any absolutely characteristic appearance. The bismuth meal remains lodged above the stricture, and in some cases it may taper downward to a point at the end of the stricture, where it remains stationary. If, after long-continued observation, very little of the meal is found to have passed through the stricture, the lower end will have the appearance of long shreds. These cases are the grave ones, usually involving the lowest portion of the esophagus, the cardia. When the cardia is involved, the esophagus is quite apt to take on the form of a spindle or funnel. In moderately severe cases the meal passes the stricture from the beginning in separate, small, long-drawn-out pieces. In the milder cases, which are mostly supra-cardiac, marked dilatation of the esophagus is usually not visible; the meal is detained for a time and then suddenly passes onward.

Cardiospasm.—Cardiospasm is not a rare occurrence. The treatment of this condition will be quite different from that of malignancy, therefore an accurate diagnosis is indispensable. In addition to the aid afforded by the esophagoscope, we can readily differentiate an organic stricture from a cardiospasm by the roentgenogram. In mild cases the esophagus above the obstruction may be dilated to twice its normal size. In cardiospasm the obstruction is funnel-shaped (Plate XI, Fig. 1), while in organic stricture the lower end is round and knob-like (Plate XI, Fig. 2). In cardiospasm the fluoroscopic examination will usually show that there is an absolute closure of the lower end of the esophagus so that no drops of the barium mixture pass off into the stomach. In malignant obstruction of the lower end of the esophagus, except in the most advanced cases, there will be found to be a minute trickling of the barium

through the diminished lumen. Another important point in the differential diagnosis is that after the spasm has been observed for several moments, if the esophagus is filled up with warm water this will cause the muscular fibers to relax and the barium mixture will rush on into the stomach. In many cases of obstruction the tissues above the stricture lose their tone, and peristalsis fails.

Diverticulum of the Esophagus.—The presence of a diverticulum is demonstrated by the intensely dark, sharply defined shadow it casts (Plate XI, Figs. 3 and 4), while at the same time a metal sound introduced beyond it in the esophagus is visible upon illumination. Should a diverticulum be completely filled with food, it is of course impossible to fill it with bismuth, and the roentgenogram will not show it. In doubtful cases examination should be made after vomiting and in various postures of the body.

Deep-seated Diverticula.—These also may be demonstrated by filling with bismuth, and the demonstration confirmed by introducing the esophageal sound; or the bismuth-filled "hog casing" may be used.

Carcinoma of the Esophagus.—Roentgenography may show a narrowing of the lumen where there are no clinical signs of such a condition or any obstruction to the passage of sounds. Irregular, jagged contours of the bismuth shadow point to the presence of carcinoma (Plate XII, Figs. 1 and 2). We are able, after filling the esophagus with bismuth, to determine in advanced cases the length of the stricture and the presence of a number of stenosed points. Constant stenosis as distinguished from spastic stenosis is of diagnostic importance. By artificially closing the lower end of the esophagus, the bismuth may be made to show slight irregularities above, which might otherwise escape notice.

EXAMINATION OF THE STOMACH.

In order to interpret the visualization of the stomach it is necessary to recognize the normal stomach and to be able to differentiate reflex from directly pathologic forms. Reflex manifestations are of a spastic character and are found in diseases of the duodenum, appendix, gall bladder, and other more remote organs. The hypodermic injection of atropin will often cause these spasms to disappear and is accordingly helpful in differentiating reflex from direct manifestations of disease.

It is difficult to describe the appearance of the normal stomach as visualized by the fluoroscopic screen. The reason for this consists in the fact that the position of the stomach varies in different individuals in accord with the bodily habitus. After one has acquired experience by the examination of a large number of individuals, he is able to predict the position of the stomach by looking at the patient's general habitus. Individuals of the spare type with flat

abdominal wall usually possess a vertical type of stomach. This vertical type in general shows the form of a gigantic letter J. The most dependent portion of the lower curve may or may not be above the intercostal line.¹ (Plate XII, Figs. 3 and 4.)

In individuals of more robust physique, with well developed muscular abdominal wall, the stomach will generally be found to be *curvée*. In those of plethoric habit with thick abdominal wall, the stomach will assume more the horizontal type. The exhaustive studies of Mills have definitely decided the above point.

Under the fluoroscope the peristaltic waves of the stomach are seen to be rhythmical in character. Normally one wave appears at a time. The presence of two or more waves gives evidence of hypermotility and, if not due to an effort to overcome an obstruction at the pylorus, may be significant of an irritation which usually has its origin in the duodenum, the appendix, or the gall bladder. In the vertical type of stomach the walls of the stomach are parallel if there is a good muscular tonus. Often during an examination this muscular tonus may be present at the beginning but under the stress of supporting the heavy meal the muscle fibers relax and we have the appearance of an hour-glass (the so-called pseudo-hourglass) with the walls of the middle portion in apparent contact. In addition to the collapse of the walls, we find a sagging of the lower portion of the stomach so that the lower pole appears very much dilated.

In gastric ulcer, only one peristaltic wave is apparent. This condition may often be differentiated from the normal by the presence of an incisura due to the ulcer.

Gastropptosis.—After the bismuth meal the stomach in gastropptosis shows as an elongated tube standing vertically to the left of the median and with its lower border far below the intercostal line. At its lower extremity it turns to the right with a sharp curve upward so that the lesser curvature shows an acute angle at the turning point; this is called a water-trap stomach (Plate XIII, Fig. 1). In some cases of gastropptosis the stomach may extend almost to the symphysis (Plate XIII, Fig. 2); the pylorus is always lower than normal. Many individuals will be found who show a rather marked gastropptosis without presenting clinical evidence. The important point to determine in these cases is whether the motor power of the stomach is competent, for if the musculature of the stomach possesses the necessary tone and strength to cause the stomach to empty in a normal period of time, there will be no clinical symptoms.

¹ The earlier method of comparing the position of the stomach with the umbilicus had the disadvantage of an inconstant factor, namely, the varying position of the umbilicus. It is accordingly more convenient to record the position of the dependent portion of the stomach by referring its relative position to a line drawn from the crest of one thumb to the crest of the other, an imaginary landmark which is known as the intercostal line.

Tonus.—Gastric tonus is the resiliency or continuous contractility of the living stomach which enables it to hold its contents firmly and to keep its own normal shape. The tonus of the stomach is demonstrated by the strength with which the organ clutches its contents. In a condition of normal gastric tonus a small quantity of fluid will almost completely fill the stomach (Plate XIII, Fig. 3), so that the upper level of the fluid is even with the diaphragm and the air bubble is in the fundus (magenblase); the walls of the stomach are vertical and parallel. Loss of tonus is accurately shown by roentgenography.

Motility.—The motor function of the normal stomach is measured by observing peristalsis and determining the time required for expulsion of the gastric contents. Peristalsis begins immediately after the ingestion of the bismuth meal, in the form of waves originating approximately in the middle of the body of the stomach and progressing toward the pylorus. They are shallow at first, but become constantly deeper. If the peristaltic waves are slow in appearing they may often be stimulated and consequently visualized by energetic manipulation of the lower pole of the stomach. The time of expulsion is determined by roentgenologically illuminating the stomach at regular intervals, beginning with the second hour after ingestion of a bismuth test meal and continuing until the last remnant of bismuth has disappeared. The normal time of expulsion is between four and six hours. Failure of the stomach to empty itself of the entire bismuth meal in four to six hours is an indication of a pathologic process, but on account of the fact that the mechanism of the stomach is oftentimes influenced by extraneous impressions, as fright, the observation should be repeated before the final conclusion is arrived at.

Atony.—A lowering of the tonus of the stomach is shown by lessened contraction around its contents, and by increased expansibility, with markedly flaccid walls. The bismuth test meal fills only the lower half of the stomach, its upper level being far below the diaphragm, perhaps only slightly above the umbilicus. Above it there is a large club-shaped air bubble (Plate XIII, Fig. 4). The stomach is narrowest at the border-line between air bubble and bismuth.

Dilatation.—In a markedly dilated stomach (benign stenosis of the pylorus) the bismuth meal fills merely the lowest portion of the stomach in the form of a broad crescent-shaped shadow, situated below the umbilicus (Plate XIV, Fig. 2). The form of the shadow is fairly horizontal; at the same time it extends far into the right half of the body. The pars pylorica is usually not well differentiated. Above the bismuth meal will be found the fluid and the stagnating food remnants which are always present in an ectatic stomach. The presence of a layer of fluid over the bismuth meal can be demonstrated by the administration of floating bismuth capsules. The

stomach increases in size if more be added to its contents. Visible remnants of bismuth twenty-four hours after the bismuth test meal are proof usually of an organic lesion at the pylorus or more frequently a lesion in the first portion of the duodenum.

An extraordinarily intensified and increased peristalsis can be observed on the screen. This is succeeded by a flaccid condition of the stomach walls due to a tiring of the muscle fibers. A reversed peristalsis, flat waves from right to left, has also been observed.

Gastric Ulcer.—A simple non-complicated ulcer cannot with certainty be demonstrated by means of the Roentgen ray. An ulcer causes a localized tonic spasm of the circular muscle fibers, inducing an incisura or deep notch on the curvature opposite. To visualize this, roentgenoscopic palpation may be necessary. The Roentgen fluoroscope may show a stoppage of the peristaltic wave at the seat of the ulcer. Hypomotility with six-hour residue is suggestive of ulcer. If the peristaltic waves are seen to traverse the lower pole of the stomach, causing indentations on the greater curvature and not on the lesser, ulcer may be suspected.

We can classify the findings in gastric ulcer under the two general headings of direct and indirect, the direct being a demonstration of a definite change resulting from the ulceration, and the indirect the disturbances in function. The most common direct findings are the bismuth fleck representing the ulcer crater, the filling defect in the gastric outline, and the organic deformities. The indirect findings are spastic manifestations, abnormalities in peristaltic waves, disturbed motility, unusual filling of the duodenum, and pressure pain points.

Callous Ulcer.—If an induration of connective tissue occurs in the vicinity of a chronic ulcer, so that the ulcer presents a crater-like opening, this crater can be filled up with bismuth and may then be seen as a small diverticular continuation of the shadow of the stomach. There is no air bubble in this ulcer cavity filled with bismuth. Induration of an ulcer of the stomach induces irregularities or absence of the peristaltic waves. Gastric ulcer, wherever located, tends to induce retention of the stomach contents. The presence of a callous ulcer may also be assumed if a persistent hour-glass stomach can be demonstrated. (Plate XV, Fig. 2.)

Perforating Ulcer.—A callous ulcer that is adherent to an adjacent organ may burrow deeply into it. The most frequent adhesions of the stomach are with the pancreas and the liver. Pockets of varying sizes are thus formed. It is a simple matter to demonstrate with the Roentgen ray the presence of such pockets. The bismuth meal partly fills the pocket, which communicates with the stomach through a narrower passage, and the cavity then appears as a diverticular attachment of the stomach shadow. This diverticulum is partly filled with bismuth (Plate XIV, Figs. 3 and 4) over which there is an air bubble (Haudek's niche). The bismuth in the pocket

sometimes appears entirely separated from the stomach shadow, without any visible connection with it. Characteristic of perforating ulcer is the fact that the shadow of the bismuth-filled pocket remains after the stomach is emptied (Plate XV, Fig. 1).

Hour-glass Stomach.—An hour-glass stomach is either functional, due to local spastic contraction (intermittent hour-glass stomach), or organic, due to cicatrices of ulcers, callous ulcers, perigastritis, or carcinomata (persistent hour-glass stomach). But mixed forms also may occur. In the persistent organic hour-glass stomach (Plate XV, Fig. 2), after the bismuth test meal, only the cardiac half of the stomach appears filled. The filling of the pyloric half may take place very gradually. If the pyloric portion fills immediately after the meal, the case is not one of organic hour-glass stomach, but may be the spastic or mixed form, which is not a rare occurrence in ulcer. Atropin may be useful in the differentiation.

Carcinoma.—The chief and frequently the only finding in gastric carcinoma is a filling defect. Depending upon the location, there will be disturbances in motility. With a carcinoma involving the cardiac end of the stomach, there is usually early emptying. In carcinoma involving the middle pole the emptying time is frequently not disturbed; and in carcinoma of the pylorus there is usually obstruction. Besides establishing the diagnosis of carcinoma, the Roentgen ray has the additional value of demonstrating the exact location of the lesion and determines the decision as to whether the case presents operative possibilities.

If the bismuth shadow shows fixed irregularities it suggests circumscribed carcinoma which projects tumor-like into the stomach cavity. This filling defect produces an irregularity of contour and narrowing of the gastric lumen. Non-projecting carcinomata which infiltrate the wall of the stomach exhibit these openings in the bismuth shadow to a much less degree, but they display a fairly strong wall, absence of peristalsis, and an area of immobility. (Plate XIV, Fig. 1.) In cases of thickening of the stomach wall a marked decrease of the lumen is observed. The most diversified roentgenograms present themselves, according to the seat, the extent, and the diffuse or circumscribed growth of the carcinoma. (Plate XV, Figs. 3 and 4.) It should be borne in mind, however, that an extragastric tumor or cyst which compresses the stomach may occasionally simulate a carcinoma of the stomach in the roentgenogram. Extra-gastric pressure, however, usually presents a smooth outline which is different from the nodular, irregular outline of carcinoma.

Pyloric Obstruction.—When the shadow near the pylorus is round and blunt and no bismuth passes through into the duodenum, a pyloric obstruction is present. (Plate XVI, Fig. 1.) Pylorospasm will simulate this condition, and serial roentgenograms may be necessary for differentiation. Pylorospasm is a constant accom-

perment of pyloric and duodenal ulcer. This spasm may usually be treated by the administration of belladonna or atropin. Delay in emptying of the stomach, associated with dilatation, indicates an obstruction of the pylorus.

The period of expulsion of the stomach contents is materially shortened by external application of heat. This fact is important in differentiating pylorospasm from organic stenosis of the pylorus. It explains also the relief of pain in the stomach by heat which relaxes the spasm of the pylorus.

The interpretation of the findings in pylorospasm and gastric retention should not be made without carefully considering the possibility of these findings being the result of a gastric manifestation of tabes. Not infrequently syphilis is the etiologic factor in pylorospasm and retention. The question of organic deformities of the stomach due to syphilis is of great importance. Undoubtedly a number of cases presenting a tumor of the stomach have been regarded as carcinoma when in reality the deformities were the result of syphilis. The roentgenologist should always suggest the necessity of differentiating between carcinoma and syphilis by other methods of examination.

EXAMINATION OF THE INTESTINE.

Roentgen-ray examination of the intestine is made by means of either the bismuth test meal or the barium sulphate enema. It is of the greatest importance that the intestine be previously evacuated by cathartics and by a cleansing enema. The administration of the cathartics should be so timed, however, that the peristaltic tone will not be affected by them when the Roentgen-ray examination is made.

Roentgen-ray examination accomplishes least in the diagnosis of conditions in the small intestine, in which only the bismuth test meal must be used. The bismuth rapidly spreads over a very large surface and casts a characteristic shadow by which the duodenum, jejunum and ileum can be differentiated. Characteristic of stenosis of the small intestine is the occurrence of a double level of the liquid bismuth-mucine. But if such roentgenograms are produced, distinct clinical signs of stenosis are already present.

Duodenal Ulcer.—Recognition of the first ascending portion of the duodenum is all-important in the roentgenographic diagnosis of duodenal ulcer. The normal filling of the bulbous duodeni with the bismuth mixture has been called the duodenal cap, from its likeness to a bishop's cap (Plate XVI, Fig. 2.) The diagnosis of duodenal ulcer by roentgenography is based on a constant deformity of this cap (Plate XVI, Figs. 3 and 4), caused by induration or cicatricial contraction surrounding the ulcer, or by spasm associated with a small ulcer. Several roentgenograms are necessary in order to

show the continuous presence of this deformity, for occasionally spasm of the cap occurs in vagotonia (see page 388). This latter condition can be completely relieved by the administration of atropin or extract of belladonna. It has been found that in duodenal ulcer the position or form of the stomach is not normal; there is either gastropnoia, or the transverse diameter of the stomach is enlarged. This latter condition is due to ultradextroposition of the pylorus, probably caused by adhesions between it and the gall bladder. Gastric peristalsis is very active in ulcer of the duodenum and is usually fairly characteristic. The waves are multiple and usually quite deep, indicating marked efforts in the propulsion of the stomach contents. The musculature of the stomach may become tired and the period of hyperperistalsis may be succeeded by a period of rest. At the same time the normal fissure between the antrum pylori and the duodenum disappears temporarily. Toward the close of the stomach-emptying process a protracted gastric stagnation often takes place, coincident with the occurrence of pain and spasm. Six hours after taking the test meal, when the stomach should be empty, remnants of bismuth can be demonstrated in it. In cases of callous duodenal ulcer which has perforated the wall, we find a niche, with retention of bismuth; sometimes a small air bubble is visible in the niche over the bismuth remnant (Haudek's niche); a specially characteristic indication of perforating duodenal ulcer is the apparent separation of the bismuth remnant from the shadows of stomach and duodenum and its continuance after the stomach is entirely empty.

Ileal Stasis.—Normally the opaque meal will have passed into the colon in twelve hours. Any residue after this time should be classified as due to ileal stasis. The importance of ileal stasis as a factor in gastric symptoms is second only to that of the appendix. The causes of ileal stasis, as determined by the Roentgen method of examination, are: adhesions and kinking, spasm of the ileocecal sphincter, and incompetency of the ileocecal valve. Under the head of adhesions should, of course, come the atypical mesenteric bands known as Jackson's membrane (see page 561). Incompetency of the ileocecal valve is most often demonstrated by means of the barium sulphate enema; but at times, by frequent observations of a given bismuth test meal, it can be definitely shown that the cecal contents have been regurgitated into the terminal ileum. The importance of incompetency of the ileocecal valve has been overestimated. Observers of wide experience state that in fully 50 per cent. of the cases given a barium enema incompetency is shown. (Plate XIX, Fig. 2.)

Colon.—The Roentgen-ray technic is exceedingly valuable in examination of the colon. The normal state of the colon, in particular, has been largely elucidated by it. (Plate XVII, Fig. 4.)

PLATE XI

FIG 1



FIG 2

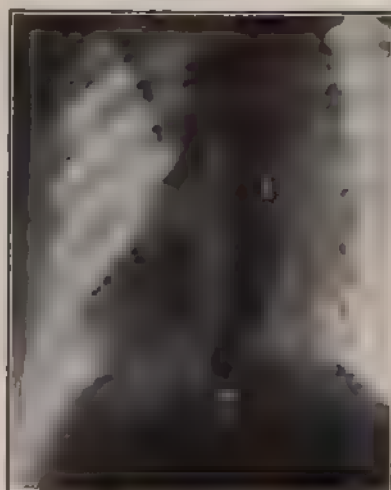


FIG. 1. CARDIOSPASM. The arrows point to the lower end of the esophagus closed by spasm. Above is seen the enormously dilated lumen of the lower esophagus. The clinical history extended over a period of several years. The diagnosis of cardiospasm was not made by the Roentgen examination. The patient was symptomatically relieved by dilatation with the water bag dilator. (P. M. Hickey and W. A. Evans.)

FIG. 2. CARCINOMA OF THE CARDIA. A, carinomatous obstruction at lower portion of esophagus. B, slightly dilated esophagus above. (P. M. Hickey.)

FIG 3

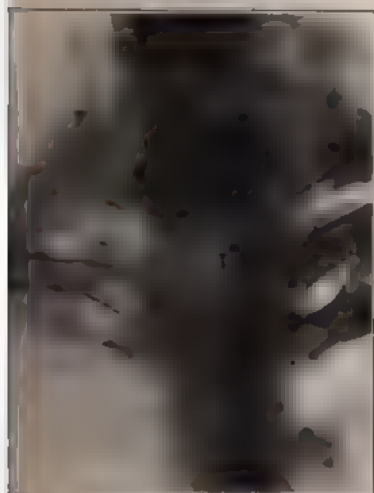


FIG 4



FIG. 3. DIVERTICULUM OF THE UPPER ESOPHAGUS. The barium is seen faintly outlined in the lower esophagus. The large rounded shadow is the ventral diverticulum with a large diverticulum. The barium mixture has overflowed and is escaping into the esophagus above. (P. M. Hickey and W. A. Evans.)

FIG. 4. LARGE DIVERTICULUM. Wide dilatation of the sac. Patient refused operation and died of inanition. (P. M. Hickey.)

PLATE XII

FIG. 1

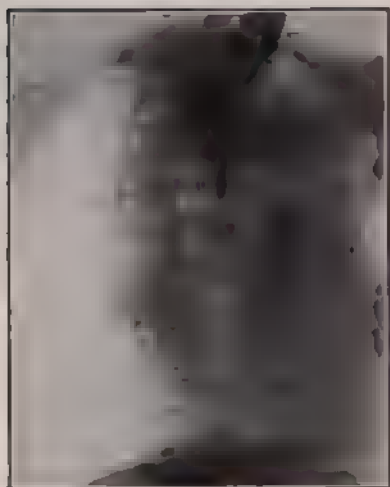


FIG. 2

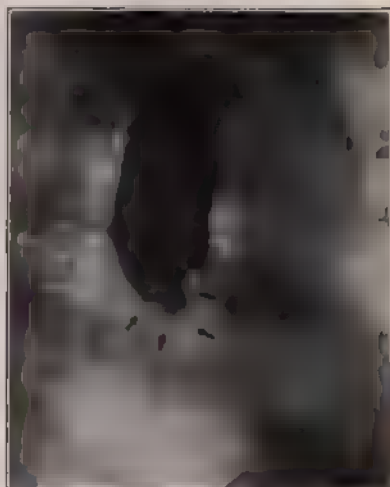


FIG. 1—CARCINOMA OF MIDDLE THIRD OF THE ESOPHAGUS. *A*, slight dilatation of the esophagus above the constriction; *B*, irregular and contracted appearance of the bismuth column, due to the encroachment of the carcinomatous mass upon the lumen of the esophagus. (P. M. Hickey.)

FIG. 2—CARCINOMA OF THE LOWER THIRD OF THE ESOPHAGUS. Note the irregular outline of the lower portion of the visualized esophagus and the marked dilatation above. The arrows point to the irregularity of the barium outline produced by the nodular condition of the new growth of the esophagus. (P. M. Hickey and W. A. Evans.)

FIG. 3



FIG. 4



FIG. 3—NORMAL HORN-SHAPED STOMACH (COW-HORN) AS FOUND IN PERSONS OF THE ROBUST TYPE. *A*, corpus; *B*, pars pylorica; *C*, pylorus; *D*, duodenal cap. (P. M. Hickey.)

FIG. 4—NORMAL FISH-HOOK STOMACH AS FOUND IN PERSONS OF THE ANTHENIC TYPE. *A*, air bubble in fundus; *B*, umbilicus; *C*, pylorus. (P. M. Hickey.)

PLATE XIII

FIG. 1



FIG. 2

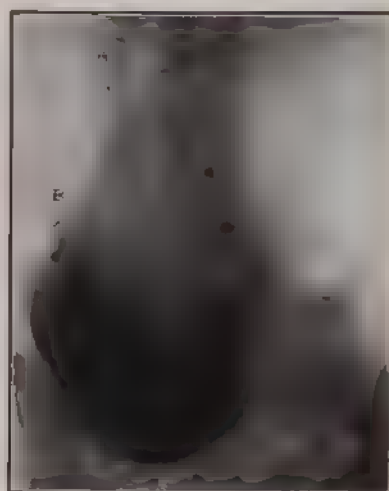


FIG. 1. GASTROPTOSIS (WATER-TRAP STOMACH). A, air bubble in fundus, B, rugæ, C, lower border of stomach, D, pylorus. (P. M. Hickey.)

FIG. 2. MARKED GASTROPTOSIS (STOMACH NEAR SYMPHYSIS). A, elongated air bubble, B, rugæ, C, lower border of stomach. (P. M. Hickey.)

FIG. 3



FIG. 4

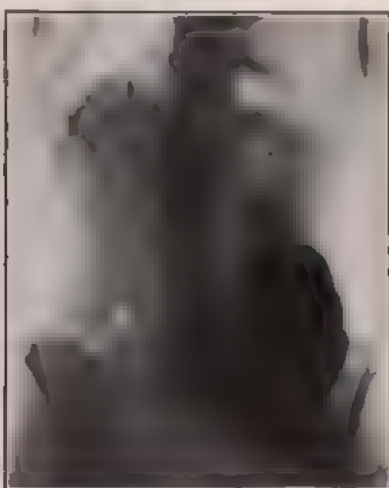


FIG. 3. NORMAL LOOPS. A, air bubble in fundus, B and C, parallel walls of the stomach, D, peristaltic waves. (P. M. Hickey.)

FIG. 4. ASYMMETRY. A, conspicuous air bubble in fundus, B, puckering of the walls of the stomach, C, dilated condition of body of stomach. Note the transverse colon. D, air below the position of the stomach. (P. M. Hickey.)

PLATE XIV

FIG. 1



FIG. 2



FIG. 1—CARCINOMA OF THE STOMACH. *A*, fundus of the stomach; *B*, deep filling defect due to the projection of the neoplasm into the stomach; *C*, drawing in of the stomach due to cicatrization of an old ulcer; *D*, pylorus; *E*, dilated duodenum. (P. M. Hickey.)

FIG. 2—SIMPLE DILATATION OF THE STOMACH. No clinical history except continual over-eating. (P. M. Hickey.)

FIG. 3

and

FIG. 4

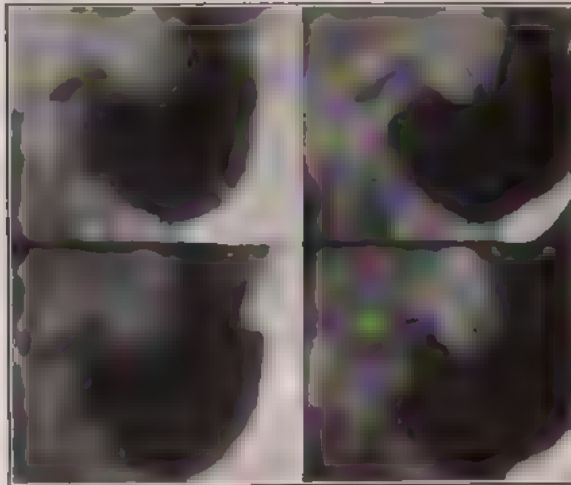


FIG. 3 and 4—PERFORATING ULCER OF THE STOMACH. Four serial plates illustrating the constancy of the lesion despite the change in the pyloric contour due to the peristaltic waves. Duodenum distorted by adhesions. Operation—excision of ulcer and gastroenterostomy. (P. M. Hickey and W. A. Evans.)

PLATE XV

FIG 1

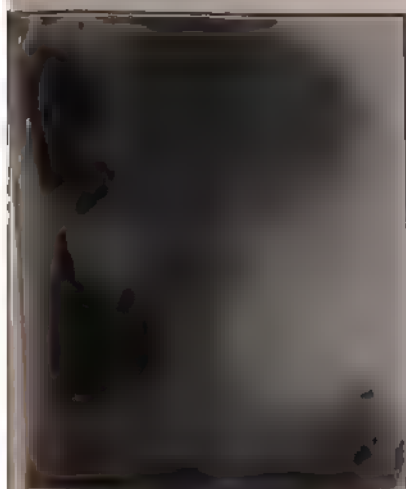


FIG 2



FIG 1. PERFORATING OF THE STOMACH. A, air bubble in fundus, B and C, middle ulcer, D, pocket due to perforation with the walls adherent, E, narrow isthmus connecting D with lower portion of stomach, F. Verified by operation. (P. M. Hickey.)

FIG 2. HOT-GLASS STOMACH. A, upper half of stomach connected by B, narrow isthmus resulting from contraction of middle of stomach due to ulcer, with cicatrization of lower pole of stomach, the pyloric portion of which is completely filled while the rest of the stomach shows the bismuth gradually trickling down through isthmus, B. (P. M. Hickey.)

FIG 3



FIG 4



FIG 3. CARCINOMA OF THE STOMACH. Infiltration of the lower pole of the stomach with a large, irregular mass. Operation. This should not be mistaken for a benign defect. This case was operative because the growth was not so extensive as to preclude operation. Compare with Plate XVI Fig 1. (P. M. Hickey.)

FIG 4. CARCINOMA OF THE STOMACH. Advanced. On account of the neoplastic mass the lower third of the stomach is inoperable. (P. M. Hickey.)

PLATE XVI

FIG 1

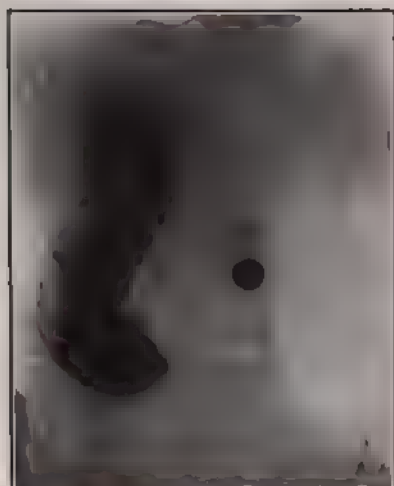


FIG 1. CARCINOMA OF THE STOMACH. Extensive infiltration extending from the pylorus along the lower border, with slight chance for escape of bismuth along the lesser curvature. A. (P. M. Hickey.)

FIG 2



FIG 2. NORMAL DUODENAL CAP. A, pylorus, B, duodenal cap—bulbus duodeni. (P. M. Hickey.)

FIG 3



FIG 3. THICKENED AND DEFORMED CAP. The incomplete filling of the upper portion of the duodenal cap was a constant finding on a series of eight plates. Operation. (P. M. Hickey.)

FIG 4



FIG 4. CHRONIC DUODENAL ULCER. Dilated stomach due to obstruction at pylorus (A, which was found at operation to be involved in a dense mass of adhesions, with chronic ulcer of the duodenum. (P. M. Hickey.)

PLATE XVII

FIG 1

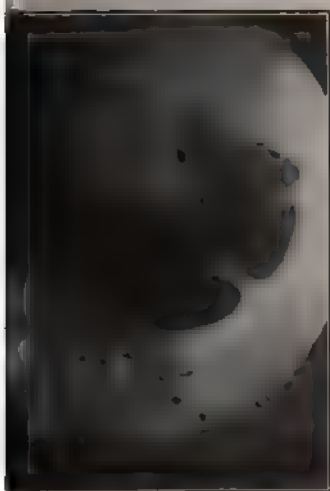
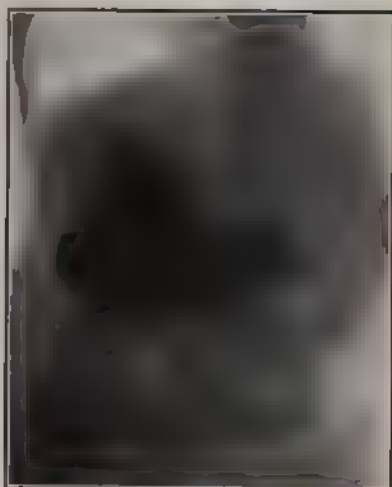


FIG 2



1. ENORMOUS DILATATION OF DUODENAL CAP. A, pylorus B, duodenal cap. Fixing on series of twelve plates. (P. M. Hickey.)

2. DILATED DUODENUM. Dilatation of second portion of duodenum due to band at embryonic origin. Operation. (P. M. Hickey.)

FIG 3

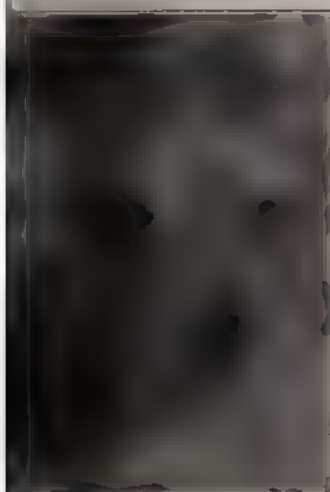
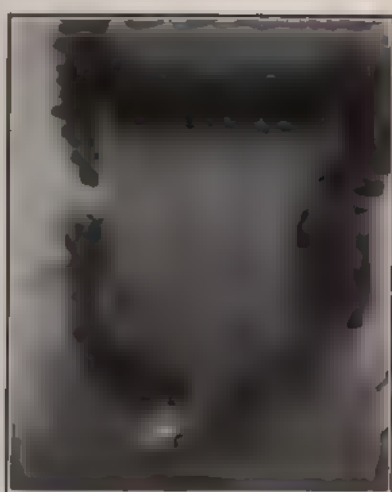


FIG 4



3. CARCINOMA SMALL INTESTINE. Marked dilatation of the small bowel. Visible through the intestine content through the distention with air. The dilatation was due to carcinoma causing the obstruction. The atria seen in the dilated small bowel are characteristic of this condition. (P. M. Hickey and W. A. Evans.)

4. NORMAL COLON. (P. M. Hickey.)

PLATE XVIII

FIG. 1

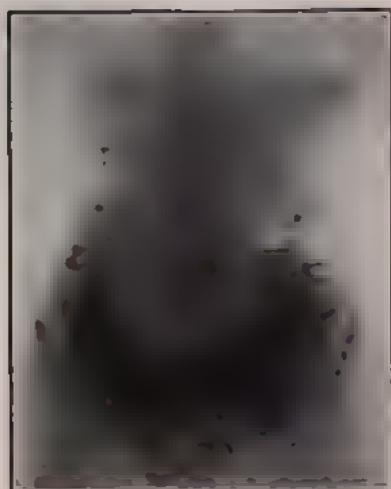


FIG. 2



FIG. 1—Coloproctosis. *A*, umbilicus; *B*, cecum; *C*, air-filled hepatic flexure; *D*, air-filled splenic flexure; *E*, transverse colon; *F*, sigmoid. (P. M. Hickey.)

FIG. 2—Acute Splenic Flexure. *A*, cecum; *B*, partly air-filled hepatic flexure; *C*, splenic flexure with left portion of transverse colon coming up and forming an acute angle with descending colon; *D*. (P. M. Hickey.)

FIG. 3

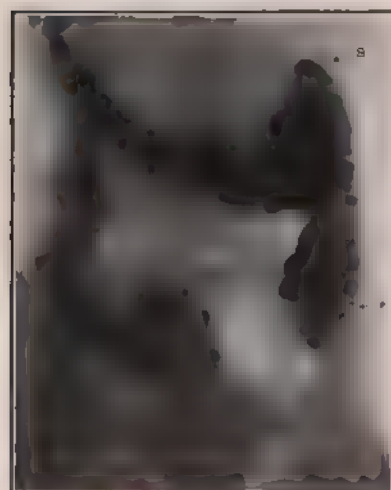


FIG. 4

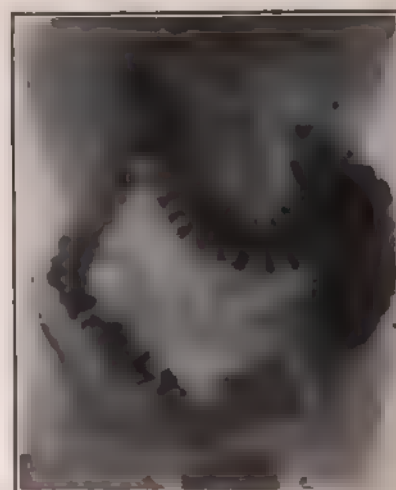


FIG. 3—Acute Hepatic Flexure. *A*, cecum; *B*, hepatic flexure the right half of the transverse colon forming an acute angle with the ascending colon; *C*, sigmoid; *D*, dilated ampulla of the rectum. (P. M. Hickey.)

FIG. 4—Normal Cecum. *A*, cecum; *B*, splenic flexure; *C*, transverse colon; *D*, hepatic flexure; *E*, descending colon; *F*, sigmoid; *G*, rectal valve. (P. M. Hickey.)

PLATE XIX

FIG. 1

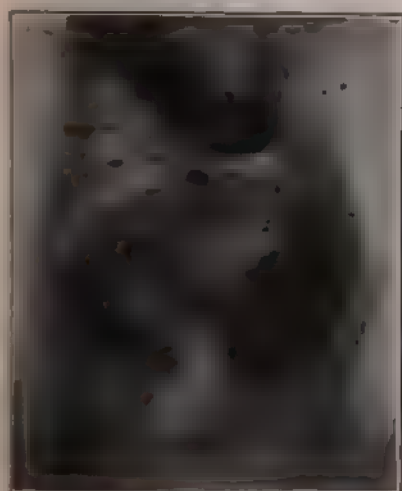


FIG. 2

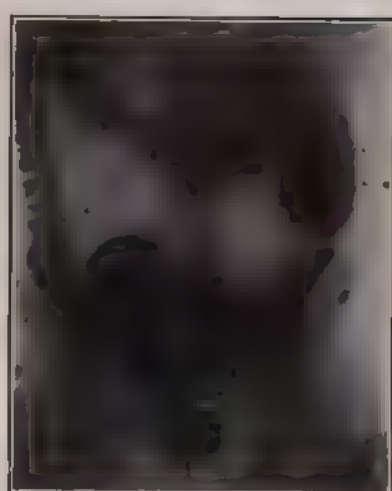


FIG. 1. PROCTOSCOPY. A, enormously dilated cecum extending well down into pelvis; B, hepatic flexure; C, splenic flexure; D, pylorus. (P. M. Hickey.)

FIG. 2. INSUFFICIENCY OF THE ILEOCECAL VALVE. A, cecum; B, ileum filled by escape of barium through cecum; C, normal sigmoid. (P. M. Hickey.)

FIG. 3



FIG. 4



FIG. 3. APPENDIX VISUALIZED THROUGH RETENTION OF BARIUM CONTENTS AS SEEN EIGHT HOURS AFTER THE INGESTION OF THE OPAQUE MEAL. The irregular filling of appendix is suggestive of partial stenosis. (P. M. Hickey and W. A. Evans.)

FIG. 4. ADHERENT SIGMOID. A, cecum; B, hepatic flexure; C, right half of the transverse colon; D, left lateral half of the transverse colon; E, sigmoid held to the right of the median line by adhesions; F, sigmoid bulb of sigmoid. (P. M. Hickey.)

PLATE XX

FIG. 1



FIG. 2



FIG. 1—CARCINOMA OF THE HEPATIC FLEXURE. Palpable mass on the right side. Patient fifty-five years of age. Constant filling defect in the hepatic flexure as outlined by barium per mouth and by barium enema. Confirmed by operation. (P. M. Hickey and W. A. Evans.)

FIG. 2—SPASTIC CONSTIPATION. A, cecum; B, hepatic flexure; C, transverse colon; D, descending colon, third day after ingestion of bismuth. Patient relieved by atropin. (P. M. Hickey.)

FIG. 3

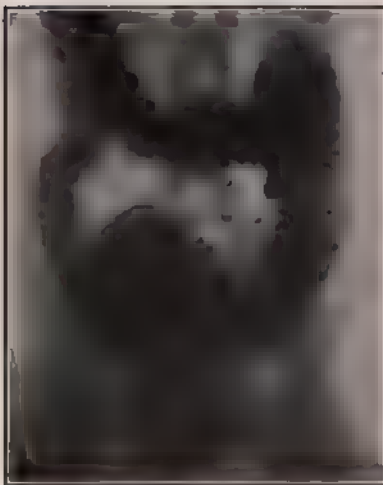


FIG. 4



FIG. 3—HYPERTROPHIED RECTAL VALVE. A, deep indentation in rectum due to hypertrophy of first rectal valve. (P. M. Hickey.)

FIG. 4—DYSCHESIA, RECTAL CONSTIPATION. A, dilated ampulla of rectum; B, hypertrophied rectal valve; C, sigmoid. (P. M. Hickey.)

PLATE XXI

FIG. 1



FIG. 2



FIG. 1—HINSCHBERT'S DILEMA. *A*, cecum partly compressed by sigmoid, *B*, hepatic flexure, *C*, transverse colon, *D*, splenic flexure, *E*, descending colon, *F*, sigmoid, *G*, ampulla. Patient referred for examination by the Juvenile Court, where he had been named as an incorrigible. (P. M. Hickey.)

FIG. 2—PERIDUODENAL ADHESIONS. Indistinct and incomplete filling of the first portion of the duodenum, due to extraduodenal adhesions. (P. M. Hickey.)

FIG. 3



FIG. 4

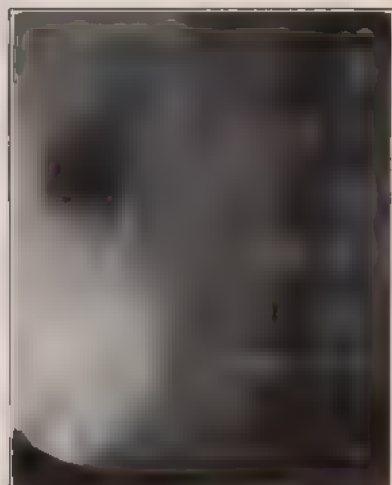


FIG. 3—GALLSTONES. Verified by operation. (P. M. Hickey.)

FIG. 4—A gallstone of such size that it was seen on the preliminary fluoroscopic exam. (P. M. Hickey and W. A. Evans.)

PLATE XXII

FIG. 1



FIG. 2

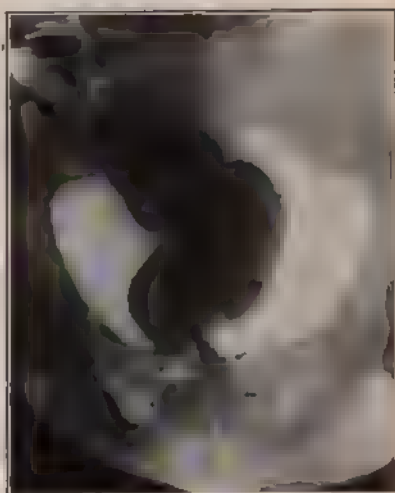


FIG. 1.—MULTIPLE DIVERTICULA. Patient complained of lower left quadrant pain for several years. On the fifth day after the ingestion of the barium meal the diverticula were quite sharply shown in the area where the patient complained of pain. The visualized appendix can also be made out in the lower right quadrant, but was found to be freely movable and not painful on pressure. (P. M. Hickey and W. A. Evans.)

FIG. 2.—CARCINOMA OF THE RECTUM. Diminished lumen of the ampulla of the rectum as visualized by barium enema. Above it to be seen the outline of the sigmoid. (P. M. Hickey and W. A. Evans.)

FIG. 3

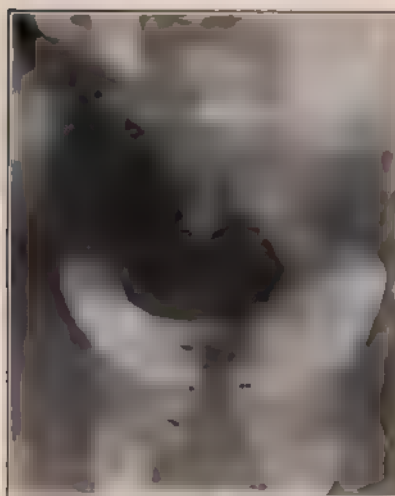


FIG. 4

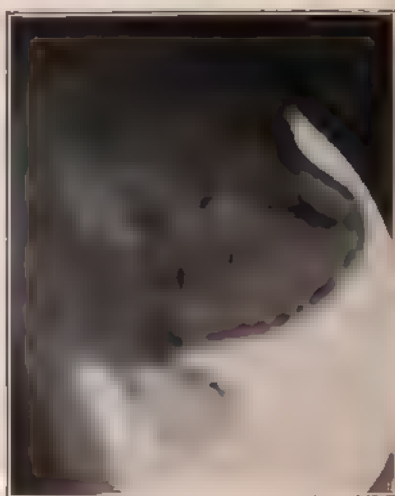


FIG. 3.—SYPHILITIC STENOSIS OF THE RECTUM. (P. M. Hickey and W. A. Evans.)

FIG. 4.—SALIVARY CALCULI. A, mylohyoid groove, B, calculus embedded in salivary duct. (P. M. Hickey.)

Normal Motility.—The first signs of shadow formation are found in the ileocecal region about four and one-half hours after ingestion of the bismuth test meal. The hepatic flexure is reached in five to eight hours; the splenic flexure in seven to fourteen hours. The final discharge of the bismuth through the anus depends essentially upon the time of defecation; at the earliest it takes place seven to eight hours after ingestion of the test meal. It is of importance to know that remnants of bismuth may normally remain in the cecum and in the ascending colon, and also in the flexures, for some length of time after the bulk of the meal has passed through these parts.

In the cecum and the ascending colon, where the opaque mass is of a relatively fluid consistency, the shadow is comparatively diffuse and indistinct. In the transverse colon and the descending colon there appear, as a result of scybala formation, sharply defined spot-like shadows, so that, in keeping with the haustral sections, a rosary-like picture of these intestinal parts is formed. (Plate XVIII, Fig. 4.) The cecum under normal conditions is situated in the right iliac fossa and continues upward, without any sharply defined demarcation, into the ascending colon. (Plate XVIII, Fig. 4.) The hepatic flexure almost never extends higher than the lower border of the right costal arch. From here the transverse colon rises in a varying bow-line to the splenic flexure, which regularly lies higher than the hepatic flexure and often touches the left diaphragm. A more or less considerable accumulation of air is generally observed in the flexures, so that it is not always easy to distinguish between a good-sized air bubble in the splenic flexure and the air bubble in the stomach. The descending colon extends vertically downward to the left iliac fossa. The sigmoid flexure shows a greatly varying loop formation.

In about 50 per cent. of patients examined by the Roentgen ray it is possible to visualize the vermiform appendix. (Plate XIX, Fig. 3.)

Position.—An exact determination of the normal position of the colon is important if effective massage of this portion of the intestinal canal is to be undertaken—for stimulation of peristalsis by massage can be accomplished only when the massage is applied to the colon in the direction of its course. (Plate XVII, Fig. 4.)

The colon is best studied after the barium sulphate enema. It is thus possible to demonstrate position anomalies, formation of loops, dilatation and stenosis of the lumen, kinking, adhesions, spasms, and palpable tumors referable to the intestine. When the lumen is obstructed it is possible to distinguish with some degree of certainty intestinal tumors, spasms, and constrictions due to adhesions. When adhesions cause kinks, distortion, displacement, or immobility of parts of the colon, they are often demonstrable. In a few cases it is possible to make an early positive diagnosis of carcinoma; in other cases the suspected stenosis or tumor can be positively located;

in others again the cause of chronic constipation is revealed. (Plate XX, Fig. 1.)

Deviation.—The greatest variety of deviations from the normal location of the colon may be observed, though it may not be possible in every instance to deduce a pathologic state from such deviation.

The most frequent deviation of the transverse colon is downward (Plate XVIII, Fig. 1). Very marked ptosis (coloptosis) is occasionally found in persons with normal intestinal function. Marked ptosis of the transverse colon is apt to bend the splenic flexure at an angle which appears in the roentgenogram to be acute. (Plate XVIII, Fig. 2.) These cases should be studied laterally or stereoscopically.

Chronic constipation and ptosis of the transverse colon are frequently associated, but the causal connection, if any, between the two conditions has not yet been made clear. On the one hand, it is quite conceivable that the transverse colon in constipated persons is dragged down by the stagnating contents of the intestine; on the other hand, however, it may well be imagined that a greatly depressed transverse colon may lead to constipation, especially if the splenic flexure is acutely bent. (Plate XVIII, Figs. 1, 2, and 3.) In the ascending-colon type of constipation the entrance of the bismuth mass into the cecum takes place within the normal time—three and one-half to five hours; but the mass remains remarkably long in the cecum and in the lower portion of the ascending colon, so that sometimes after twenty-four hours, or even longer, little or none of the bismuth has reached the transverse colon. The bulk of the mass may be gradually moved along, large remnants remaining in the cecum for a considerable length of time.

In a second group of cases characterized by ptosis and marked loop formation of the transverse colon, the passage of the bismuth mass through the entire colon is uniformly retarded. In these cases the colon in its entire length seems to be able to cast a shadow for a long time (up to forty hours). (Plate XIX, Fig. 1.)

A third group includes cases of spastic constipation with hypotonicity of the lower portion of the colon and normal or decreased tonicity of the upper portions. (Plate XX, Fig. 2.) The lower portions appear narrower than the upper.

A fourth group is composed of so-called dyschezia (Plate XX, Fig. 4), or rectal constipation (Hertz). In these cases the bismuth is retained for days in the sigmoid flexure and in the rectum, while the upper portions of the colon are emptied in the proper time. Much information may be gained by roentgenography in regard to the size and action of the rectal valves. (Plate XX, Fig. 3.)

Cecum Mobile.—The pathologic changes of position of the cecum include also the mobile cecum described by Wilms, which is readily diagnosed by means of the Roentgen ray in the alternating dorsal and lateral positions (see page 770).

Volulus of the cecum has also been successfully diagnosed with the aid of the Roentgen ray, the intestine being filled with bismuth. It has been found that in infiltrating, indurating and ulcerating processes in the cecum and in the ascending colon the contents of the intestine pass the affected portions so quickly that the bismuth casts no shadow between the lower ileum and the transverse colon.

Appendix.—As already stated, the vermiform appendix can be retained in about 50 per cent. of cases. A diagnosis of chronic appendicitis may be made when, first, the appendix is visualized and the pain point is seen directly over the shadow of the bismuth-filled appendix; second, when the cecum is not easily movable by external manipulation; and third, when the appendix and lower portion of the cecum do not empty at the same rate as the rest of the ascending colon.

The importance of the appendix in gastro-intestinal disease cannot be overstated. The Roentgen evidences of appendiceal disease are, for the most part, direct. The most common are (a) retention—the degree of retention usually determining the importance of the appendix as a factor in the gastro-intestinal symptoms; (b) tenderness localized to the appendix; (c) kinking or angulation of the appendix, indicating that appendiceal drainage would be imperfect; (d) irregular filling, suggesting either contractions or constrictions; (e) adhesions; (f) position; (g) incompetence of the ileocecal valve. (Plate XIX, Fig. 3.)

The most frequent indirect sign of appendicitis, or even pathology in the right lower quadrant, is the so-called right-sided position of the stomach. We are often able to suggest on the first gastric study that the condition is one of right lower quadrant, pathology from the fact that the stomach is drawn downward and far to the right.

In cases where the appendix cannot be seen, one is justified in suggesting a diagnosis of appendicitis if there is tenderness of the cecum on deep pressure, and if there is cecal fixation and retention, or cecal spasm. When manipulation of the cecum or pressure over the appendiceal region produces pain in the epigastrium in the absence of other disturbances, appendicitis is probably present (see Barons sign, page 773).

Colonic Stasis.—In connection with the colon, the most important condition for the internist to consider is colonic stasis. The most common cause of constipation, as shown by the Roentgen examination, is involvement of the pelvic colon in adhesions. Aside from the fixation of the bowel and tenderness associated with manipulation of the part, spasticity of the pelvic colon is always suggestive of adhesions. The various deformities of the cecum, cecal fixation, and sharp angulations at the flexures are also associated with disturbed colonic motility. The diagnosis of carcinoma or other new growths involving the colon is facilitated by the demonstration of a definite defect in bowel outline or by an obstruction produced by the involvement of the lumen with the tumor.

Sigmoid Flexure.—Aside from the transverse colon, the most pronounced deviations occur in the course and in the position of the sigmoid flexure. (Plate XIX, Fig. 4.)

Roentgen-ray examination may be of value in these cases in determining or excluding adhesions. Especially suitable for this purpose is the Roentgen fluoroscope; during examination the positions of the different loops as compared with one another and their motility toward each other may be rendered clear by pushing them apart with either the protected or the gloved hands or Holzkecht's "detector."

Diverticulitis.—Hernial protrusions develop at the mesenteric attachment between the layers of the mesentery. (See pages 785, 786 and 787.) When these protrusions become inflamed we have diverticulitis. Their most common location is at the sigmoid flexure. By means of the Roentgen ray they can now be easily demonstrated. (See Plate XXII, Fig. 1.)

Hirschsprung's Disease.—This congenital dilatation of the lowest portions of the colon and rectum can be easily recognized by means of the Roentgen ray. (Plate XXI, Fig. 1.)

Stenosis. An enema is most suitable for Roentgen-ray examination for stenoses. The patient should assume the dorsal position on the examination table. The short intestinal tube connected with the irrigator is introduced into the rectum, the bismuth clysma is slowly injected, and the entrance and advance of the fluid are observed on the screen from the first moment until it reaches the cecum. At the same time the abdomen may be palpated and massaged with the hand, and the intestinal loops pushed apart in order to obtain greater clearness. The haustral segmentation may be absent when the enema is given. Under normal conditions the entire colon is filled, up to the cecum, in a few minutes, and for this purpose one liter of fluid is usually sufficient. Insufficiency of the ileocecal valve may occasionally be determined by entrance of the bismuth into the lowest portion of the ileum. (Plate XIX, Fig. 2.)

In cases of intestinal stenosis the advance of the bismuth column is halted, according to the degree of the stenosis, for a longer or shorter period. If the stenosis is complete the shadow ends funnel-shaped or broadly, and the filling of the upper portions of the intestine is impossible; or if not quite complete, the pointed or broad ending of the bismuth sends toward the upper parts a narrower, often irregularly defined, split-up shadow. It is true that in stenoses which are not very marked and which clinically do not as yet manifest any distinct symptoms, the bismuth column is also retarded for a little while, but then the upper portions of the colon are gradually filled in a normal manner, so that between the normally filled intestinal parts which are situated above and those situated below the stenosis a distinct absence of the shadow is visible, corresponding to the stenosed part. It is often necessary, for an exact

diagnosis, to make the roentgenographic examinations at different times, especially when there is a question of excluding spastic stenoses. A stenosis may also be visible on the Roentgen fluoroscope when the bismuth enema is allowed to run out through the rectal tube, the space below the stenosis being emptied quickly, whereas the portion above remains filled with bismuth and is emptied only very slowly. This method of examination is to be applied also in cases of stenosis due to adhesions and flexures of the intestine. To localize and determine the degree of a stenosis, various authors recommend the use of hardened gelatin or glutoid capsules of different sizes filled with bismuth, some of which remain lodged at the stenosis; but this method has not found general acceptance. (Plate XXII, Fig. 3.)

The diagnostic importance of the shadows cast by regions containing air or water cannot be overestimated. Some authors regard these shadows as pathognomonic of stenosis.

Diagnosis of Postoperative Obstructions.—A Roentgen-ray examination in postoperative obstruction of the bowel is necessary in every case before further operative measures are employed. By this procedure an acute dilatation of the stomach is immediately recognized. The roentgenograms reveal gas distention in the small or large intestine, which may be easily distinguished by the characteristic outlines of the gas areas.

Pancreas.—The study of the pancreas is rendered difficult both by its structure and by its relations. Well developed cysts of the head of the pancreas have been recognized during a Roentgen examination, by the displacement and change in the relations of the pylorus and duodenum. Carcinoma of the pancreas has also been diagnosed by the disturbance in outline and relations of the duodenum produced by its presence.

Liver.—The indications for roentgenologic study of the liver are limited. An enlargement of the liver is shown sometimes from distortion of the diaphragm line or displacement of the abdominal contents.

Gall Bladder.—For several years the study of the gall bladder by Roentgen examination was confined to the demonstration of calculi and adhesions. Until recently no routine examination of the gall-bladder region was made, the question of adhesions being determined during the study of the duodenum. It has been customary to describe the so-called gall-bladder position of the duodenum in its relation to the pylorus, and also to explain certain deformities of the duodenum by assuming periduodenal adhesions complicating a cholecystitis. When the duodenum was shown toward the median line and somewhat upward, when the pylorus extended a little too far to the right, when the mobility of the duodenum was reduced, and when tenderness accompanied manipulation of the duodenum, we have assumed the existence of gall-

bladder disease. The pathologic conditions demonstrated have included hydrops of the gall bladder, empyema of the gall bladder, and chronic thickening of the gall-bladder wall. Inasmuch as the normal gall bladder is rarely demonstrated, we can safely assume that a shadow of this organ definitely indicates pathology.

Gallstones.—Roentgenographically about 50 per cent. of gallstones can be diagnosed. Pure cholesterol stones absorb the ray to a slight extent and are therefore rarely detected on the plate. When there is a deposit of $2\frac{1}{2}$ per cent. or more of calcium the shadows of the gallstones are readily seen. (Plate XXI, Figs. 3 and 4.) The smaller the quantity of bile in the gall bladder, the more easily can gallstones be demonstrated. When there are periduodenal or perigastric adhesions due to cholecystitis the stomach and duodenal cap assume a characteristic position (Plate XXI, Fig. 3). The presence of gall-bladder disease can often be surmised from the appearance of the bismuth-filled stomach. If adhesions are present, due to chronic inflammation of the gall bladder, the pyloric portion of the stomach is usually pulled well over to the right, extending oftentimes two or three inches beyond the median line. The appearance of the duodenal cap in these cases is also characteristic. If the adhesions do not involve the duodenal cap sufficiently to distort it, we will find that the cap makes an acute angle with the stomach so that the peak of the cap is drawn upward and to the left. This abnormal position of the stomach to the right of the median line, and the angulation of the cap, constitute the so-called gall-bladder position of the stomach, and may indicate a pathologic condition of the biliary ducts or gall bladder. In plates of the gall-bladder region, a much thickened gall bladder will often show a distinct outline, due to the fact that the infiltrated mucosa casts a differentiating shadow.

Spleen.—The differential diagnosis of tumors in the upper left quadrant can be aided by the demonstration of the splenic outline. In order to show this organ, it is necessary to distend the stomach with gas, and also to have considerable liquid in the stomach. With the patient on the right side, with the above conditions complied with, the splenic outline is frequently very well shown.

Peritoneal Inflation.—The injection of oxygen into the peritoneal cavity is one of the newer procedures which bids fair to make possible many diagnoses now considered difficult. Enough oxygen is injected into the free peritoneal cavity to form a background of lessened density against which the visceral organs are graphically displayed. In this way the outline of the liver, the size, shape and position of the spleen, the presence or absence of adhesions, and the easier demonstration of gallstones, with visualization of the outline of the gall bladder, will be realized.

CHAPTER VI.

DIET IN GASTRIC DISEASES.

FOR practical purposes food may be defined as any substance which, when taken into the body, assists in its nutrition and maintenance, or replaces its waste and losses. Food has two main functions—namely, the provision for growth and repair of the animal body, and as a source of potential energy to be converted into heat and work. Substances which may not serve either of these functions may yet fulfil a useful place in a dietary. Such articles as tea, coffee, and meat extractives, while they cannot be properly classed as foods, are important, nevertheless, in the consideration of dietetics.

Food as it is ingested differs widely in its composition from the nutrient material ultimately required for the repair of waste and the sustenance of the body. Before it can be utilized in the animal economy it must undergo a more or less complex process, designated "digestion," which means alteration in the alimentary tract by certain unorganized ferments (enzymes).

Diet plays the most important part in the treatment of diseases of the stomach. In prescribing diet for patients with gastric disease of any kind, great care should be exercised to avoid that which will tend to irritate the affected stomach. A properly selected diet usually fulfils a number of indications, such as diminution of the production of mucus, or increased or decreased secretion of acid; it will obviate the danger of overburdening the muscular coats, and in this way fortify the tone of the stomach. Reduction of abnormal fermentative processes may be accomplished by a properly selected diet.

The progress made in the treatment of diseases of the stomach and intestine has been due mainly to more accurate knowledge of the chemical composition of food and of the changes that take place within the human organism. Simple methods of examining the stomach contents have inclined physicians to make greater use of the stomach tube to ascertain qualitative and quantitative deviations from the normal in gastric digestion. The results obtained by accurate analysis render the prescribing of proper diet a comparatively easy matter. Since we are able to remove and examine the stomach contents at will, we can adapt the treatment to the disease much better than would be possible if dietetic directions had to be given without the laboratory aids.

Heat Value of Foods.—The heat values of the various foodstuffs have been determined by experiment, and the result is expressed in calories. A calorie is the amount of heat required to raise the

temperature of one kilogram of water 1° C., or approximately the amount required to raise the temperature of one pound of water 4° F. According to Atwater, 1 gram of protein furnishes 4 calories, 1 pound 1820; 1 gram of fat furnishes 9 calories, and 1 pound 4004; 1 gram of carbohydrate furnishes 4 calories, and 1 pound 1820. The caloric value of foods must be borne in mind. Patients suffering from gastric disease are usually placed on a too restricted diet; the number of caloric units is too small, and as a consequence the patients rapidly lose flesh. It is absolutely necessary in all cases of chronic disease of the stomach to see that the patient obtains the required number of calories every twenty-four hours.

Fat in the form of butter is one of the best foods for developing heat without injuring the stomach. In all chronic diseases of the stomach, fat agrees well. In many diseases of the digestive organs the most satisfactory progress has been made by adding great quantities of fat to the dietary. Investigations in metabolism have verified this conclusion.

Dietary Regulations and Lists.—The experience which the patient has gained in reference to his own diet should be taken into consideration when prescribing a diet for him. The postulate of Boas, "throw away the printed dietary lists," is based upon the desire to escape from monotonous routine in the treatment of patients suffering from gastric disorders, inasmuch as it is not possible to satisfy the subjective sensations of the patients by means of fixed rules. Patients frequently maintain that they are unable to digest certain articles of food. Such assertions vary, but they correspond to the peculiar nature of gastric digestion, inasmuch as the assimilability of certain articles of food differs markedly in different patients. The habits of the patient are likewise to be taken into consideration. The preference for or objection to certain foods, the desire for change or for certain modes of preparation, the behavior of the patient as to appetite and the sensation of hunger, are all of great importance when considering the selection of a menu. Appetite and hunger are trustworthy guides to the healthy man for the food requirements of the body. In health, as much food as the normal appetite calls for is generally eaten; this corresponds, as a rule, to the quantity which can be assimilated and by which the body weight is kept fairly constant for a considerable space of time. In patients with disease of the stomach the appetite and the sensation of hunger are, as a rule, not a trustworthy guide for the quantity of food required; in most cases both are below normal.

When this is the case, the diet must be regulated in such a manner that nutrition does not suffer on account of the deficient appetite. To diet does not mean to starve. It is also of great importance to search for the causes of the anorexia. These may consist of organic disease of the stomach, or they may be of a purely nervous

AVERAGE COMPOSITION OF COMMON AMERICAN FOOD PRODUCTS
(AT WATER).

Food materials (as purchased)	Refuse	Water	Protein	Fat	Carbo- hy- drates	Ash	Fuel value per pound.
	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent	Calo- ries
ANIMAL FOOD							
Beef, fresh							
Chuck ribs	18.3	52.6	15.5	15.0		0.8	910
Flank	10.2	54.0	17.0	19.0		0.7	1025
Lean	13.3	52.5	16.1	17.5		0.9	1025
Packerhouse steak	12.7	52.4	19.1	17.9		0.8	1100
Section steak	12.8	53.0	16.5	16.1		0.9	975
Neck	27.0	45.9	14.5	11.9		0.7	1185
Ribs	20.6	43.5	13.0	21.2		0.7	1135
Roast rolls		63.9	19.3	16.7		0.9	1055
Round	7.2	60.7	19.0	12.8		1.0	890
Ham	20.7	45.0	13.8	20.2		0.7	1090
Shoulder	36.9	42.9	12.8	7.3		0.6	345
Shoulder and head	16.4	50.8	16.4	9.8		0.9	715
Forequarter	18.7	49.1	14.5	17.5		0.7	905
Ham, quarter	15.7	50.4	15.4	18.3		0.7	1045
Beef, canned, canned, pickled, and dried							
Canned beef	8.4	40.2	14.3	23.8		4.0	1245
Tongue, pickled	6.0	58.0	11.9	19.7		4.3	1010
Dried, salted, and smoked	4.7	53.7	20.4	6.9		8.9	790
Canned beef		51.8	25.6	22.5		1.3	1410
Canned corned beef		51.8	26.3	18.7		4.0	1370
Lamb							
Breast	21.3	52.0	15.4	11.0		0.8	745
Leg	14.2	60.1	15.5	7.9		0.9	625
Leg, outside	3.4	68.8	20.1	7.5		1.0	695
Forequarter	24.5	54.2	15.1	6.0		0.7	535
Ham, quarter	20.7	50.2	16.2	6.6		0.8	580
Mutton							
Ham	9.9	30.0	14.8	36.9		0.8	1770
Leg, hind	18.4	51.2	15.1	14.7		0.8	860
Leg, chops	16.0	42.0	13.5	28.3		0.7	1415
Forequarter	21.2	41.6	12.3	24.5		0.7	1245
Ham, quarter, without tallow	17.2	45.4	13.8	23.2		0.7	1210
Pork							
Breast	19.1	45.5	15.4	19.1		0.8	1075
Leg, hind	17.4	52.9	15.9	13.6		0.9	860
Pork, fresh							
Ham	10.7	48.0	13.5	25.9		0.8	1320
Shoulder	19.7	41.8	13.4	24.2		0.9	1245
Shoulder	12.4	44.9	12.0	20.8		0.7	1460
Shoulder, ham		66.5	18.9	13.0		1.0	895
Pork, salted, cured, and pickled							
Ham, smoked	13.6	34.8	14.2	33.4		4.2	1635
Shoulder, smoked	18.2	36.8	15.0	26.6		5.5	1335
Ham, pick		7.9	1.9	80.2		3.9	3555
Shoulder, smoked	7.7	17.4	9.1	62.2		4.1	2715
Poultry							
Chicken	3.3	35.2	18.2	19.7		3.8	1155
Duck		39.8	13.0	44.2		2.2	2075
Geese, broiler		57.3	19.0	18.0		1.1	1155
Geese							
Geese, green of		88.0	2.1	2.8	5.0	1.5	235
Geese		92.9	4.4	4.3	1.1	1.2	120
Geese, green of		81.5	4.6	4.3	5.5	1.1	305
Geese		90.0	1.8	1.1	2.6	1.5	185
Geese, broiler							
Geese, broiler	41.6	43.7	12.8	1.4		0.7	305
Geese	23.9	47.1	13.7	12.3		0.7	785
Geese	17.6	38.5	13.4	20.8		0.7	1475
Geese	22.7	42.4	16.1	18.4		0.8	1060
Geese							
Geese, dressed	29.9	55.5	11.1	2.2		0.8	220
Geese, broiler of sections	17.7	61.9	15.3	4.4		0.9	475
Geese, broiler	44.7	40.4	16.2	4.2		0.7	370
Geese, yellow, dressed	35.1	50.7	12.8	7.7		0.9	275
Geese, white	50.1	35.2	9.4	4.8		0.7	380
Geese		71.2	20.9	3.8	2.6	1.6	600
Geese, broiler							
Geese, broiler	24.9	40.2	16.0	4.1		15.5	325
Geese, broiler	41.4	19.2	20.5	8.5		7.4	755

* Refuse, oil.

AVERAGE COMPOSITION OF COMMON AMERICAN FOOD PRODUCTS
(ATWATER)—continued.

Food materials (as purchased).	Refuse.	Water.	Protein.	Fat.	Carbohy- drates.	Ash.
ANIMAL FOOD—continued.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Fish, canned:						
Salmon	5.0 ¹	63.5	21.3	12.1	2.6
Sardines	5.0 ¹	53.6	22.7	12.1	5.3
Shellfish:						
Oysters, "solids"	55.1	6.0	1.3	2.3	1.1
Clams	80.3	10.6	1.1	5.2	2.3
Crabs	62.4	26.7	7.9	6	1.5
Lobsters	81.7	20.7	5.9	2	.8
Eggs: Hens' eggs	11.3 ²	65.8	12.19
Dairy products, etc.:						
Butter	11.0	1.0	85.0	3.0
Whole milk	87.0	4.0	8.0	.7
Skim milk	90.5	3.4	5.1	.7
Buttermilk	91.0	1.0	5	4.8	.7
Condensed milk	26.9	54.1
Cream	74.0	3.5	4.6	.5
Cheese, Cheddar	27.4	27.7	86.8	4.1	4.0
Cheese, full cream	34.2	25.9	2.4	3.0
VEGETABLE FOOD.						
Flour, meal, etc.:						
Entire-wheat flour	12.4	13.8	1.9	71.9	1.0
Graham flour	11.3	15.1	2.2	71.4	1.0
Wheat flour, patent roller process:						
High-grade and medium	12.0	11.4	1.0	75.1	.8
Low grade	12.0	14.0	1.9	71.2	.9
Macaroni, vermicelli, etc.	10.3	13.4	.9	74.1	1.3
Wheat breakfast food	9.6	12.1	1.1	75.2	1.1
Buckwheat flour	13.6	6.4	1.2	77.9	.7
Rye flour	12.99	78.7
Corn meal	12.5	9.2	1.9	75.4	1.1
Oat breakfast food	7.7	16.7	7.3	66.2	3.1
Rice	12.3	8.0	.3	79.0	.4
Tapioca	11.4	.4	.1	88.0	.1
Starch	90.0
Bread, pastry, etc.:						
White bread	35.3	9.2	1.3	54.1	1.1
Brown bread	43.6	5.4	1.8	47.1	3.1
Graham bread	13.1	8.9	1.3	54.1	1.6
Whole-wheat bread	25.4	9.7	49.7
Rye bread	25.7	9.0	.6	53.7	1.6
Cake	19.9	55.5
Cream crackers	6.8	9.7	12.1	69.7	1.7
Oyster crackers	4.8	11.3	10.5	70.5	2.0
Soda crackers	5.9	9.8	9.1	73.1	2.1
Sugars, etc.:						
Molasses	70.0
Candy ³	96.0
Honey	81.0
Sugar, granulated	100.0
Maple syrup	71.4
Vegetables: ⁴						
Beans, dried	12.6	22.5	1.8	55.5	3.5
Beans, Lima, shelled	68.5	7.1	.7	22.0	1.7
Beans, string	7.0	83.0	2.1	.3	6.9	.7
Beets	15.0	10.0	1.3	.1	7.7	.9
Cabbage	15.0	77.7	1.4	.2	4.8	.5
Celery	20.0	75.6	.9	.1	2.6	.7
Corn, green (sweet), edible portion	75.4	3.1	1.1	19.7	.7
Cucumbers	15.0	81.1	.7	.2	2.6	.4
Lettuce	15.0	80.5	1.0	.2	2.5	1.3
Mushrooms	3.5	.4	6.8	1.3
Onions	10.0	78.9	1.4	.3
Parsnips	20.0	66.4	1.3	.4	10.8	1.1

¹ Refuse, shell.

² Plain confectionery not containing nuts, fruit, or chocolate.

³ Such vegetables as potatoes, squash, beets, etc., have a certain amount of inedible skin, seeds, etc. The amount varies with the method of preparing the vegetables, and is accurately estimated. The figures given for refuse of vegetables, fruits, etc., are as represent approximately the amount in these foods as ordinarily prepared.

COMPOSITION OF COMMON AMERICAN FOOD PRODUCTS 155

AVERAGE COMPOSITION OF COMMON AMERICAN FOOD PRODUCTS
(AT WATER)—continued.

Food materials (as purchased).	Refuse.	Water.	Protein.	Fat.	Carbohy- drates.	Ash.	Fuel value per pound.
VEGETABLE FOOD—continued.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Calo- ries.
Vegetables—continued.							
Peas, <i>Pisum sativum</i> , dried	9.5	83.3	2.5	1.0	62.0	0.8	1565
Peas, <i>Pisum sativum</i> , shelled	74.6	7.0	.5	.5	16.9	1.0	440
C. peas, dried	13.0	21.4	1.4	1.4	60.8	3.4	1505
Potatoes	20.0	62.6	1.6	.1	14.7	.8	295
Rhubarb	40.0	56.6	.4	.4	2.2	.4	93
New potatoes	20.0	55.2	1.4	.6	21.9	0	440
Spinach	92.3	2.1	.3	.3	3.2	2.1	95
Beetroot	46.2	7	.2	.2	4.5	.4	169
Tomatoes	94.3	0	.4	.4	3.9	.5	100
Turnips	30.0	62.7	.9	.1	5.7	.6	120
Vegetables, canned.							
Jacked beans	68.9	2.5	10.6	2.1	555		
Peas, <i>Pisum sativum</i> , green	53.3	3.4	2	1.1	235		
Corn, green	74.1	2.8	1.2	10.0	9		430
Beetroot, green	75.0	3.0	1.0	18.6	.9		425
Tomatoes	94.0	1.2	.3	4.0	.6		95
FRUITS, berries, etc., fresh.							
Apples	25.0	63.3	3	3	10.8	.3	100
Oranges	35.0	48.9	8	.4	14.3	.6	200
Grapes	25.0	58.0	1.0	1.3	14.4	.4	295
Lemons	30.0	62.6	7	.5	5.9	.4	125
Muskmelons	30.0	44.8	.3	.4	4.6	.3	80
Strawberries	27.0	63.4	.6	.4	8.5	.4	180
Pears	10.0	76.0	.5	.4	12.7	.4	230
Prunums, edible portion	66.1	.8	.7	31.5	.6		550
Raspberries	85.8	1.0	12.6	.6	280		
Strawberries	5.0	85.9	.9	.6	7.0	.6	150
Watermelons	59.4	37.6	.2	.1	2.7	.1	50
FRUITS—dried.							
Apples	26.1	1.6	2.2	66.1	3.0		1185
Apricots	29.4	4.7	1.0	62.5	2.4		1125
Dates	13.8	1.9	3.6	70.6	1.2		1275
Figs	18.8	4.3	.3	74.2	2.4		1280
Prunes	13.1	2.3	3.0	68.6	3.1		1205
Nuts.							
Almonds	45.0	2.7	11.5	58.2	0.8	1.1	1515
Brazil nuts	49.6	2.6	8.0	33.7	3.5	3.0	1485
Butternuts	50.4	3.6	3.8	8.3	5	.4	385
Chestnuts, fresh	16.0	37.8	5.2	4.8	35.4	1.1	915
Chestnuts, dried	24.0	4.5	8.1	6.3	56.4	1.7	1385
Cocconuts	45.8	7.2	2.9	25.9	14.3	.9	1295
Cocconuts, prepared	53.1	3.5	6.3	57.4	31.8	1.3	2605
Pistachios	52.1	1.8	7.5	31.3	6.2	1.1	1440
Pineapples	62.2	1.4	5.8	25.5	4.3	.8	1145
Peanuts	55.2	1.4	5.2	33.2	5.2	.7	1405
Walnuts	24.5	6.9	19.6	29.1	18.5	1.5	1775
Walnuts, black	40.6	2.0	9.7	34.8	10.2	1.7	1730
Walnuts, English	71.1	.6	7.2	14.6	3.0	.5	730
Macadamias	58.1	1.0	6.9	30.6	6.8	.6	1250
Miscellaneous.							
Chocolate	5.9	12.9	48.7	30.3	2.2		2025
Cocoa, powdered	4.0	21.6	28.9	37.7	7.2		2100
Cereal coffee infusion (1 part boiled in 30 parts water) ¹	96.2	.2			1.4	.2	30

¹ Fruits contain a certain proportion of inedible materials, as skin, seeds, etc., which are properly classed as refuse. In some fruits, as oranges and pears, the amount rejected in eating is practically the same as refuse. In others, as apples and peaches, more or less of the edible material is and nearly rejected with the skin and seeds and other inedible portions. The edible material which is thus thrown away and should properly be classed with the waste, is here classed with the refuse. The figures for refuse here given represent, as nearly as can be ascertained, the quantities actually reported.

² Milk and shell.

³ The average of five analyses of cereal coffee grain is: Water, 6.2; protein, 13.3; fat, 3.4; carbohydrates 72.0 and ash, 4.5 per cent. Only a portion of the nutrients, however, enters into the infusion. The average in the table represents the available nutrients in the beverage. Infusions of genuine coffee and of tea like the above contain practically no nutrients.

nature. The diminished appetite may have been induced artificially by a dietary plan which the patient himself had determined upon before seeking medical advice. The statements of patients regarding the digestibility and general effects of certain articles of food must not be accepted unreservedly as a guide to treatment.

Average Composition of Common American Food Products (Atwater).

—The table on pages 153, 154 and 155, compiled by Atwater, taken from Bulletin No. 142 of the United States Department of Agriculture, gives the average composition of ordinary American food materials, with the percentage of refuse.

Digestibility is a term which is frequently misunderstood and misapplied. Digestibility does not involve the question of distress, nor the question of the food containing sufficient calories; for not every food which is well digested is well borne, and not every food which is well borne is digested well and assimilated properly. The amount of digestive effort required varies with different articles of diet. A person whose gastro-intestinal tract is normal can digest and assimilate without discomfort any kind of reasonable food which he can eat.

The term "digestible" signifies, however, something quite different in gastric disease. Riegel defines a diet as easily digestible which does not make great demand on the secretory and motor functions of the stomach, and which is easily absorbed without producing subjective discomforts. This definition also includes assimilability, or good effect after absorption. Wegele claims that a food is easily digestible when it fulfils the following conditions: (1) It must offer but little resistance to the digestive juices; that is, it must be easily soluble. (2) It must not impede the peristaltic movements, nor, on the other hand, should it accelerate the movements of the stomach too much. (3) It must not seriously irritate the digestive organs chemically or mechanically. (4) It must be easy of absorption, either from the stomach or from the intestine.

Experience teaches that emaciation and loss of strength must be referred, in the majority of cases of gastric disease, to insufficient nutrition. It is therefore important to know the quantity of food absolutely needed by the body. In health the requirements for the average adult are 100 grams of protein, 50 grams of fat, and 450 grams of carbohydrate, daily. Expressed in calories, the number is 2720. Individual articles of diet may be substituted one for another in proportion to their heat values. Obviously the constitution and the appetite of the patient offer certain limits which must be respected. If one or more ordinarily desirable articles of diet are not acceptable to the patient, we may substitute others, so long as we supply the required number of heat units. When considering this question, it is important to remember that a person at rest requires fewer calories than one at work. In health the requirements per kilo of body weight per day, at rest, are 30 to

35 calories; at light labor, 35 to 40 calories; and at average labor, 40 to 45 calories. In the treatment of severe chronic gastric disease the patients should be confined to bed in order to economize the heat units contained in the comparatively small amount of food they are able to take.

Vitamin.—It has been proved that, besides protein, carbohydrates, fat, mineral salts, and water, another substance is essential to the maintenance of orderly metabolism and the proper nutrition of the body. This substance is called "vitamin." When vitamin is lacking, nutritional "deficiency" or "avitamin" diseases develop, extreme examples of which are beriberi, scurvy, and rickets.

As long ago as 1897 the disastrous effects of an exclusive diet of polished rice on the nutrition of fowls was discovered (Eyckman); and at the same time it was shown that fowls suffering from polyneuritis as a result of the feeding with polished rice recovered promptly under a diet of undecorticated rice. (It cannot be said even now that the birds deprived of the antineuritic vitamin to the point of prostration from polyneuritis are not permanently injured, but they respond rapidly in general condition to feeding with the rice polishings or an extract made from them, or to a diet of yeast extract.) Not only rice, but bread and milk, supplied the material for the early investigation of the presence of a previously unsuspected vital principle. By extracting a mixture of milk and bread with ether and alcohol, it was found that, while the residue contained the well known protein and other chemical ingredients of these articles, something was missing, and that mice, which can flourish on a diet of unmodified bread and milk, would perish if given no other food than the modified mixture (Stepp). The extract contained a principle analogous to that contained in the polishings of rice; for when it was fed to the starving mice they recovered their wonted health. In 1911 this vital principle was named "vitamin" by Casimir Funk. Other names have been suggested since, but all workers in the science of nutritional deficiency now employ the term "vitamin."

The presence of vitamin in milk was demonstrated by Osborne and Mendel and by Hopkins about 1910, by feeding small quantities of the milk, in its natural condition or protein-free, to animals reduced in vitality by living on "purified" proteins and other foods. The stimulating effect of the milk on such animals was out of all proportion to the known nutritive principles contained in this fluid, even in its natural, protein-bearing state. Then McCollum and Davis discovered the fact that butter and the yolk of eggs contained an ingredient that could not be classified simply as fat or oil, for it had a peculiar stimulating effect on growth, differentiating it clearly from lard and olive oil.

Meanwhile the preparation of extracts of rice polishings and other vitamin-containing substances proceeded. An efficient alcoholic extract of rich polishings was made by Fraser and Stanton in 1907;

and in 1911 Funk showed that pressed yeast hydrolyzed with sulphuric acid had the same effect as rice polishings in curing polyneuritis in birds. Thus one by one were the sources of vitamin being discovered, and methods of extraction devised. But it soon became evident that there was more than one vitamin—that the occult substances extracted from grains, egg yolk, etc., were classifiable according to their physiologic effects; that polyneuritis in birds and beriberi in man were not the only results of vitamin-deficiency in the food; and that the remedy in each instance must be related to the cause.

Among other effects of vitamin-deficiency the following have been clearly established: xerophthalmia, an edematous affection of the eyes, not curable by medicinal means or local applications, and leading ultimately to total blindness; rachitis; and scurvy.

The vitamins, not being chemically identifiable, have been grouped in two classes according to their solubility, as "fat-soluble" (soluble in fat-solvents) and "water-soluble." And it has now become necessary to subdivide the latter into two classes, called respectively "B" and "C," the fat-soluble vitamin being known as "A."

Although vitamins are present in a great variety of food substances, if the food is to be selected for vitamin purposes it is well to make the selection from among those which contain an abundance of the particular vitamin desired. The fat-soluble "A" vitamin (indicated in xerophthalmia, rickets, dental caries, and impaired bodily growth) is abundant in butter, eggs, and cod-liver oil, and fairly plentiful in cabbage, carrots, cream, milk, sweet potatoes, spinach, and whole wheat flour. Water-soluble "B" vitamin (indicated in beriberi, impaired growth, and a general condition of malnutrition) is abundant in navy beans, soy beans, sweetbread (pancreas), yeast, and whole wheat flour, and fairly plentiful in bananas, carrots, cauliflower, celery, Indian corn or whole corn meal, milk, oats, onions, parsnips, peanuts, potatoes, rutabaga, spinach, and whey. Water-soluble "C" vitamin (indicated in scurvy, actual or threatened) is abundant in apples, fresh cabbage, lettuce, onions, oranges, fresh peas, and spinach, and fairly plentiful in potatoes and tomatoes.

This list is not complete; but more important than a more extended list, perhaps, is the consideration that, though present in certain vegetables in the natural state, vitamins are influenced in varying degrees by heat and by acids and alkalis. The different classes of vitamins thus far discovered (there may be others awaiting discovery, or what are now classed under one head may be found to belong under two or more) have been grouped according to their stability with reference to temperature, acids and alkalis as follows (Drummond): "A," stable at 100° C., probably at 140° C.; "B," comparatively stable at 100° C., slowly destroyed at 120° C. and

above; "C," gradually destroyed above 50° C., rapidly destroyed above 80° C. "A," stable in cold alkali, probably stable in acids; "B," slowly destroyed in cold alkali, rapidly in hot, comparatively stable in acids; "C," rapidly destroyed even in cold alkali, comparatively stable in acids below 50° C. These calculations are not intended to be taken as anything but approximations. The "B" vitamins appear to be uninjured by long boiling with acids, and it has been claimed that the "A" type as found in butter will withstand the effect of live steam. The "C" vitamin is least resistant to extraneous influences; but, on the other hand, it is not necessarily subject to such influences when appropriated in the form of fruits—apples, oranges, and lemons. The antiscorbutic vitamin in milk is probably injured or destroyed by pasteurization; hence the desirability of giving bottle-fed babies a little orange juice, since their diet consists entirely of milk. The boiling process may injure or extract the "B" or "C" vitamins in potatoes and other vegetables, but in baking the intense heat of the oven does not penetrate to the interior. In eggs, which contain "A" vitamin mostly, the vitamin is retained despite boiling or poaching. The "C" vitamin in cabbage cannot survive boiling; to obtain it in this food, the latter must be served cold—as "slaw."

It has been shown experimentally that both the fat-soluble "A" and the water-soluble "B" vitamins are essential to normal growth—of rats, fowl, swine, etc., and inferentially of man. The water-soluble "C" vitamin is absolutely essential to health, as are the other two, but its absence is not manifested otherwise, so far as known, than by the development of scurvy.

The vitamins are not nutrients in any sense of the word. So far are they from taking the place of any of the well known constituents of a normal diet that their administration will often stimulate the appetite; in indicated cases more food than before is made physiologically available, and hence more is demanded, though the equivalent of the former ration is more completely utilized than it would be without the necessary vitamins.

The three vitamins in extract form are now obtainable for medicinal use, being marketed under the name metagen. Metagen is supplied in capsules of 0.3 Gm. (5 grains) each—one ordinary dose.

Meat.—Much discussion has taken place regarding the respective merits of light and dark meat. The significance of the distinction has frequently been exaggerated, but certain differences should not be disregarded. White meat (veal, fowl) possesses a shorter, softer, and more tender fiber, and differs from dark meat, such as beef and mutton, by its smaller proportion of extractives; this difference is considerable. The fat of dark meat is with difficulty dissolved, and therefore offers resistance to penetration by the digestive juices. Meat may be defined as the muscle of an animal together with the conjoined connective-tissue substances,

such as tendons, ligaments, bones, and cartilage. The internal organs of the animal, so far as they are edible, namely, kidneys, spleen, liver, sweetbread, brain, and intestine, as well as sea-foods, such as fish, lobster, and clam, are also included under the term "meat." The average composition of meat is: protein, 20 to 25 per cent.; fat, gelatinous substances, glycogen, and extractives (kreatin, xanthin). The meat of animals recently killed should be permitted to hang for some time before it is eaten. While meat is hanging, lactic acid is produced which loosens the connective tissue between the muscle fibers, thereby softening it and rendering it more easily digestible. Game should hang for a time to permit of this softening process, especially for patients with gastric troubles; but it should not be allowed to decompose so far as to become "gamey," for then it is likely to arouse the aversion of the patient or, if eaten, to increase the digestive disturbances by introducing into the system the products of decomposition. Old animals naturally yield tougher meat than young.

The preparation of meat is important. Raw meat ought to be avoided by patients with gastric disease, though it is more easily digested in health than is cooked meat. The digestion of raw meat takes place both in the stomach and in the small intestine; the coarse connective tissue is digested by the stomach, the muscle fiber by the small intestine. The stomach cannot, however, digest raw meat when the secretion of hydrochloric acid is defective; on the other hand, when there is too much hydrochloric acid, still more of it is produced under the stimulus of raw meat in the stomach. The dictum of Schmidt to strike out raw, rare, and smoked meat from the diet of gastric patients is therefore a reasonable one. Smoked and canned meats behave similarly toward hydrochloric acid. Salted or canned meats, such as ham, ox tongue, smoked or corned beef, have not the same nutritive value as raw meat or meat prepared in any other way; during the pickling process, extractive materials and phosphates are lost. In partaking of uncooked meats there is always a possibility of infection by animal parasites. The custom among the German people of eating raw pork is well known. Thousands of microscopists are employed in Germany to prevent trichinosis. Stiles found that of 274 cases of trichinosis in America 208 were Germans. The simplest and most effective method of preventing the disease is ignored. The cooking of the meat is all that is necessary.

The majority of people eat more meat than they require. Meat once a day is sufficient for a person not engaged in manual labor, or for one who does not take much vigorous outdoor exercise. A high blood-pressure may be more or less lowered by excluding meat from the diet. Acute rheumatic patients are better off without meat. Many gastric troubles owe their origin to the consumption of food which causes a greater drain on the gastric juices than the

system is able to stand. Of the various meats, young lean beef is, as a rule, the most easily digested. The white meat of a fowl enjoys a special reputation, to which most clinicians agree; yet no chemical differences between the white meat and dark meat have yet been shown.

The digestibility of roasted, boiled, and stewed meats is in the order named. It may be increased by preliminary processes such as beating, grinding, mincing, and scraping. Meats poor in fat are generally easily digestible, owing to the ready accessibility of the connective tissue to the gastric juice. The following varieties are permissible for gastric patients: Beef, veal, lamb, lean pork, hare, deer, fowl, squab, partridge, pheasant, and all kinds of lean fish, such as trout, pike, codfish, and shad. Because of their high percentage of fat, goose, herring and salmon should be avoided. Caviar, though rather salty, may be allowed, as may also oysters and lobsters. The meat of the lobster is not so tough and difficult to digest as is popularly believed. Sausage should be avoided because it contains about 40 per cent. of fat and a great deal of condiment.

Gelatin.—Foods containing gelatin belong to the group of meat nutrients. They are of great importance in dietetics, serving as protein and fat spacers. Gelatin is almost entirely digested, leaving little or no residue. Tendons, cartilage, ligaments, connective tissue, and bones belong to this class. Gelatin is present in larger amount in the broth of veal than in beef broth. Calf's head and calf's feet are rich in gelatin. Meat jelly is a popular gelatinous food for patients with stomach disease. Meat broth is not considered a food, since it contains only small quantities of protein, fat, and gelatin. It is, however, rich in extractives which stimulate the secretion of hydrochloric acid. The indications for its use are therefore plain. Bouillon soups containing eggs or flour may be substituted for pure meat broths.

Beef Tea.—Pure beef tea has much the same value as meat broth. It contains rather more protein and gelatinous substances than broth, and has a marked effect in stimulating the appetite, favoring the secretion of gastric juice in cases of acute and chronic affections of the stomach. Beef tea is prepared by taking fresh meat free from fat and cutting it into small pieces, placing it in a bottle without water, and slowly heating it on the water-bath; after steaming for twenty minutes the meat juice collects as a turbid, yellowish fluid. (See page 177.)

Eggs. Eggs are to be taken, as such, only when soft boiled, the white coagulated. In the form of very light egg dishes, or stirred up in soups, they make a very acceptable addition to the diet. Hard-boiled or fried eggs cannot readily be reached by the gastric juice, and are apt to irritate a diseased gastric mucous membrane, unless the hard protein is first very finely triturated.

They are valuable, however, in cases of hyperchlorhydria. Eggs are a concentrated food containing 13 per cent. of protein and 9 per cent. of fat. A diet consisting solely or chiefly of eggs should not be prescribed for patients with gastric trouble, since even in health it is not advisable to ingest large quantities of protein in so concentrated a form. If the functional derangement of the stomach is marked by subacidity, the peptonizing power is of course deficient; and if by hyperacidity, the secretion of acid will be still further augmented by the ingestion of protein. Raw eggs are not assimilated as well as boiled eggs. They frequently cause diarrhea and vomiting. It is estimated that only 50 per cent. of the whites of raw eggs is utilized in the human digestive tract. This assertion may be somewhat startling, but the recent work of Mendel, Lewis, Ely, and Bateman proves it to be true. Raw egg-white contains something, in small amount, which has a powerful retarding or inhibiting action on the digestive enzymes. This is destroyed by heat. The egg should be coagulated by heat to favor its digestion and assimilation.

Fat.—A diet rich in fat was at one time considered deleterious to patients with gastric disease. At present, however, the view predominates that fat is a food which is very well adapted to this class of patients, inasmuch as it has a high caloric value in proportion to its volume. We know, moreover, that fat hinders the secretion of gastric juice, while it does not interfere with the motility of the stomach. Good results have been reported after the administration of fat in cases of disturbed motor activity of this organ. The fat best adapted to patients with gastric disease, as has been said, is butter. There are but few trustworthy substitutes for good butter. Of other fats suitable for patients with stomach diseases, we have cream, olive oil, oil of sesame, sweet oil of almond, and cod-liver oil. Because of its disagreeable taste cod-liver oil proves repulsive to most patients; the taste may be disguised by administering the oil in capsules.

Milk.—Since milk contains large proportions of protein, fat, carbohydrate, and vitamin, it is an excellent article of diet. When a liquid diet alone is indicated, milk holds first place. It must, however, be borne in mind that milk alone is unable to supply the required number of calories, for three liters of milk contain only 1800 calories. Should milk prove repulsive to a patient, the taste may be disguised by combining it with other articles of diet. Milk ought to be given freshly boiled, as it is then more easily digested, and the germs contained in it are destroyed by the boiling process. Milk is poorly borne by many patients, and for various reasons. Sometimes the reason is purely subjective, a sort of "phobia" that has to be overcome by psychic influence. Then, again, with a pure milk diet a large quantity is necessary in order to provide the required number of calories, and this large volume interferes with the digestion of the milk. The volume may be diminished

by the use of condensed milk. The stomach contents are apt to become excessively acid when great quantities of milk are taken, the acid-secreting glands being stimulated by the presence of the milk. When it is considered advisable to prescribe milk in large amounts, the hyperacidity may be corrected by the use of alkaline mineral waters in small amounts, beginning during the second hour of gastric digestion. The addition of lime-water to milk will often aid in its digestion in cases in which the milk would not otherwise be easily borne. Milk is nearly always well borne if it does not remain too long in the stomach. The more finely the casein floccules are precipitated the less discomfort is the patient likely to experience from a milk diet. The drinking or sipping of milk in very small quantities will cause a fine precipitation of casein in the stomach. Part of the pronounced value of koumiss and kefir is due to the precipitation of casein in a finely subdivided condition. Peggins, a sterile milk-sugar rennet ferment, has a similar action in producing a finely floccular coagulation. It is found that milk causes much discomfort in cases of stenosis of the pylorus.

Karell Cure.—For certain disorders, such as dropsical conditions of all kinds, whether due to the heart, the kidneys or the liver; for asthma resulting from emphysema and pulmonary catarrh; obstinate neuralgia; hypertrophy and fatty degeneration of the liver, and all obscure conditions of the gastro-intestinal tract and the nervous system, a type of milk diet advocated by the Russian physician, Karell, has proved useful. The patient is kept on this diet until all the acute symptoms have subsided and then gradually placed on one more nutritious. This, the so-called "Karell cure," consists in limiting all foods taken by the patient to skimmed milk in amounts of two to six ounces (60 to 200 Cc.) at exact intervals, four times a day. It can be taken at any temperature preferred by the patient's taste, and should be sipped or "chewed" so that it will be intimately mixed with the saliva. If it is well digested, as proved by solid stools, the amount is gradually increased until at the end of two weeks one quart (1000 Cc.) is allowed. Karell insists upon the observance of regular intervals between the milk meals—8 o'clock, 12, 4 and 8. During the first week some difficulty is encountered, and each feeding seems a very tiny dose, yet if the patients follow the rule they complain of neither hunger nor thirst. Constipation is relieved by simple enemata. If thirst is distressing, a little plain water or seltzer is allowed. If the desire for solid food in the second or third week is overpowering, an allowance of food from the purin-free list is gradually added to the diet. Absolute rest in bed is essential. The "cure" is indicated in cases of high blood-pressure; decompensated acute and chronic heart disease associated with cyanosis, dyspnea, edema, and ascites; arteriosclerosis, kidney and liver diseases, gout, and rheumatic arthritis. In many cases a prompt

and efficient diuresis results in loss of weight and disappearance of all edema. The improvement in the subjective symptoms is often marvellous.

Should milk as such not be permissible, advantage may be taken of one or more of the numerous milk preparations available.

Buttermilk contains less fat and sugar than fresh milk, but the small percentage of lactic acid it contains gives it an agreeable and refreshing taste; it is well borne in gastric disease and is particularly useful in febrile affections of the stomach.

Whey, the fluid remaining after the precipitation of casein, contains protein, milk-sugar, peptone, and common salt; its nutritive value is small, and it is used to a very limited extent (see page 690).

Koumiss and Kefir.—Koumiss and kefir, on the other hand, are most excellent milk preparations. Koumiss is prepared from the milk of either mares or cows by lactic acid and alcoholic fermentation. It contains lactic acid, carbon dioxid, and alcohol, and has an agreeable, slightly acid taste. Kefir has been used much more extensively than koumiss. It is prepared by means of kefir tablets or pastilles, which acting upon milk produce lactic and alcoholic fermentation, the result of which is a thick, cream-like, acidulous beverage. Boiled milk, cooled, is poured over the kefir ferment and left to stand for twelve hours at ordinary room temperature. It is then stirred, filtered, and placed in bottles, which are to be thoroughly shaken three times a day and kept in a cool place. After two or three days the kefir will be ready for consumption. The advantage of kefir is that it contains small quantities of carbon dioxid together with a very small percentage (2 per cent.) of alcohol. When ingested it hastens the secretion of hydrochloric acid and, on account of the action of the carbon dioxid, increases its power.

Yoghurt. This is a Bulgarian sour milk, of recent introduction in America, but long used in the East. It has a high nutritive value, employed in the same way as kefir (see page 691).

The value of yoghurt (pronounced *yogourt*) for gastric patients was first appreciated by the Bulgarian physician Grigoroff, and later by the French school. It is similar in many ways to kefir and koumiss. The acidulation is generated by a ferment containing three kinds of bacteria, the most important of which is the *Bacillus bulgaricus*, a long bacillus which appears both singly and in chains, and which can be stained by Gram's method. This bacillus is able to induce fermentation of dextrose, sugar of milk, and saccharose, and causes the coagulation of sterile milk within twelve hours by the formation of lactic acid. A temperature of 60° to 70° C. destroys the vitality of the germ in thirty minutes. The composition of yoghurt and its relation to ordinary sour milk (which becomes acid by mere exposure to air), to kefir, and to koumiss, may be seen from the following table:

	Kefir	Koumiss	Common sour milk	Yoghurt
Lactocasein	2.68	0.80	3.55	2.70
Lacto-albumin	0.28	0.30		0.98
Peptones and albumoses	0.05	1.04		3.75
Fat	3.10	1.12	3.70	7.20
Milk-sugar	2.78	0.39	4.50	9.40
Lactic acid	0.81	0.96	0.60	0.80
Alcohol	0.70	3.19		0.20
Mineral constituents .	0.79	0.33	0.71	1.38

The advantage of yoghurt consists in the fact that its casein and albumin are rendered soluble in the shape of peptones and albumoses, and that the lime phosphates have gone into solution up to 68 per cent. These facts serve to explain the ready digestibility of the milk.

Metchnikoff ascribes a direct life-prolonging effect to yoghurt milk, and he bases this opinion upon the fact that in Bulgaria, where yoghurt is a regular article of diet, there are in four million inhabitants three thousand six hundred consumers of yoghurt who are said to be above one hundred years of age, while in Germany, with a population of sixty-one million, there are only about seventy centenarians. Granted that the conclusions of Metchnikoff may be somewhat erroneous, it must still be admitted that the decomposition process in the intestine and the whole tissue metamorphosis are favorably affected by the use of yoghurt. Preparations analogous to yoghurt are put out by the various pharmaceutical houses in America. Tablets are manufactured from a pure culture of Bulgarian lactic acid bacilli, which when added to sweet milk induce fermentation, the result being a beverage that is essentially the same as yoghurt.

Cheese.—Cheese is made by treating raw milk with rennet. The resulting coagulum is thoroughly beaten up, and then left standing to mature. The casein is thereby split up into various decomposition products which give the cheese its characteristic odor and taste. Decomposed cheese, which is looked upon as a delicacy by some people, should not be prescribed for patients with gastric disease. Almost every normal stomach rebels against the Roquefort and Limburger cheeses with their characteristic odors. The semiputrid casein cheese should never be eaten even by healthy people, not to mention people with impaired digestion.

Bread.—Rye bread is prepared from rye flour by means of yeasted dough. Black bread and "pumpernickel" are made from rye flour; Graham bread from whole wheat meal. All these varieties must be excluded from the diet of gastric patients, inasmuch as they prove a source of irritation to any but a normal stomach. The finer baked foods, especially those made of wheat, as white bread, zwieback, and cookies or biscuits prepared by the addition of butter, milk, and sugar, are especially adapted for gastric treatment. Ordinary wheat bread should be given stale, or only when

roasted as toast. Fresh and very soggy wheat bread retards penetration by the digestive fluids and is difficult of mechanical subdivision. Wheat bread toasted, zwieback, and biscuits, like the crust of bread, contain their starch in the form of dextrin which is easily digested. It is, however, necessary that patients with gastric disease should carefully masticate and insalivate these baked foods.

There is a mistaken idea among the laity that the sick should be fed pappy, liquid substances entirely different from the food taken by a person in health. If we except foods that contain a great deal of irritating waste, there is no reason why, in general, the diet of the sick should differ from that of the well. We must, however, eliminate fried foods and fermentable vegetables. Pure white bread is never contra-indicated, and the addition of butter gives it a high caloric value. The bread should be thoroughly toasted in order to dextrinize the carbohydrates and render them easily digestible. Crackers can frequently be substituted for bread.

Gruels. Grain flours are used not only in the baking of bread, but in the preparation of soups. The gruel soups in the making of which the grain granules are first boiled and then pressed through a sieve are valuable in the treatment of stomach diseases. Oat and barley gruel are prepared after this manner. Their mucoid consistency is due to gluten and broken-up starch granules. Gruel soups protect the mucous membrane of the stomach from the irritating effects of other foods eaten at the same time. Sago and tapioca as porridge or soup are very useful foods for gastric patients. Noodles, macaroni and spaghetti are useful farinaceous dishes.

Potatoes.—The cheapness of potatoes and the large percentage of carbohydrates they contain render them a very satisfactory food for all classes of gastric patients. They must be properly prepared—as a purée, if need be, with the addition of milk and butter. Other tuberous plants used as foods are much poorer in carbohydrates than potatoes, and should be eaten only when they can be prepared in the form of purée. Hard tubers, such as radishes, beet roots, and onions, are contra-indicated in cases of impaired digestion.

Rice. Rice has usually been considered an inferior food owing to the excess of starch (in other words, deficiency of protein) in its composition; and this is undoubtedly true of rice as we usually get it. This alleged defect in the grain is due to the removal of a nutrient substance in making it presentable for the market by what is known as the polishing process. Not only the outer husk, but what is known as the "rice meal," which envelops the inner kernel, is removed, despite the fact that this is the most nutritious part of the grain. Analysis of "rice meal" shows it to contain 12.5 per cent. of protein and 4.5 per cent. of phosphoric acid. The Japanese, in common with other rice-eating peoples, polish only the grain that is intended for export; what is kept for home

consumption, being unpolished, possesses a much larger proportion of nutriment and a flavor which the polished grain lacks. Rice in its natural condition is therefore a very nutritious article of food; it is easily digested, and quite suitable for patients with impaired digestion. The polished rice is so deficient in vitamin that animals fed solely upon it develop polyneuritis analogous to that which occurs in beriberi. (See page 157.)

Green Vegetables.—Green vegetables and the various kinds of cabbage contain very little protein and only a small quantity of carbohydrates. Prepared as purées they are permissible, however. The small percentage of cellulose in green vegetables is no contra-indication to their use. A patient with gastric disease should not, however, eat vegetables which cannot be finely divided. Of asparagus, the tops only are allowable. Mushrooms are contra-indicated. Green vegetable leaves are rich in vitamin (see page 158).

Legumes.—Peas, beans and lentils are all rich in protein, containing about 20 or 25 per cent., and 50 per cent. of carbohydrates. They are consequently very nutritious substances, and, when well cooked and carefully strained, suitable for gastric patients.

Fruit.—Fruit contains less protein than do vegetables, but a larger quantity of carbohydrates in the shape of dextrose and levulose. The refreshing taste of fruit is due to various fruit acids, such as malic acid in apples, tartaric acid in grapes, and citric acid in lemons. Patients with gastric disease should take fruit only when it is cooked by boiling.

Sugar.—Cane-sugar, grape-sugar, milk-sugar, or fruit-sugar may be eaten by patients with gastric disease, within certain limits. Solutions of sugar cause in the stomach a decreased secretion by the gastric glands. Since they inhibit the secretion of hydrochloric acid, they are applicable in conditions of hyperacidity. Morgan carefully experimented with cane-sugar on several persons, making repeated gastric analyses; he concluded that sugar in considerable amounts in the diet of either the healthy or the sick depresses the secretory functions of the stomach. In hyperchlorhydria a diet containing large amounts of sugar diminishes the secretion of hydrochloric acid in about the same proportion as it does in a healthy stomach. Three or four ounces of sugar can be digested by the healthy adult without difficulty in twenty-four hours. Saccharin occasionally gives rise to disturbed digestion.

Spices.—Small quantities of common salt stimulate the secretion of gastric juice; large quantities hinder digestion. The ingestion of salt in gastric disease has to be regulated according to the findings on analysis of the stomach contents. In cases of achlorhydria salt is indicated, while in cases of hyperchlorhydria it is contra-indicated. In addition to sodium chlorid, the alkaline phosphates and earths are made use of in the human economy. They are, however, present in ordinary food in sufficient quantity for this

purpose. Only very few spices should be allowed in the dietary of gastric patients. Vanilla and cinnamon are harmless. Practically all other spices must be eliminated, or used with care for the purpose of stimulating an insufficient secretion of gastric juice.

Water.—Artificial waters charged with carbon dioxid have no place in the dietary of stomach patients. The natural mineral waters, however, excite peristaltic action and have a slightly anesthetic effect upon the mucous membrane of the stomach. Strong natural waters, like the artificial substitutes, contain too much carbon dioxid, and consequently have a harmful effect upon the stomach. Not more than eight ounces of water should be taken at one time. Water is not absorbed by the stomach. The drinking of ice-water is harmful, inasmuch as it temporarily paralyzes the pyloric closure, so that the stomach contents are in danger of being emptied at once into the duodenum. As Bettmann has shown, large draughts of hot water benefit those who are well nourished and whose digestive tract is well supplied with muscular tissue. Large draughts of hot water, taken on retiring, are beneficial to corpulent people who are subject to "bilious attacks" so called, or who are affected with gastric catarrh. An aperient pill swallowed at bedtime with a large tumblerful of hot water is usually all the medicine that is necessary to keep such patients comfortable. Hot water taken before meals, either with or without phosphate or sulphate of soda, is also beneficial. It acts by dissolving and washing out of the stomach the accumulated mucus. Such treatment, however, instituted in cases of motor insufficiency (atony, dilatation), almost invariably does harm. At first the patients experience some relief, but after a few weeks all their symptoms return in an aggravated form. This is explained by the fact that when the digestive tract is relaxed and muscularly weak the stomach is unable to propel large quantities of fluid into the intestine. In such cases it is always difficult to get sufficient water into the system. The stomach should not be overloaded with water at any one time, but water should be taken in small quantities and frequently.

Recent experiments on dogs prove that the ingestion of quantities of water with food causes a marked increase both in the quantity of the gastric juice and in its hydrochloric acid content.

Alcohol.—The combustion of protein and fat is diminished after small quantities of alcohol are taken. In acting as a fat-sparer, alcohol itself is consumed and yields heat and energy to the body; it is, therefore, to a certain degree a food. Alcohol is usually consumed in the shape of champagne, beer, wine, whisky, brandy or other concentrated spirituous liquor. The general effect of alcohol in small quantities and in not too concentrated form on gastric digestion is to stimulate secretion; but large quantities and the concentrated drinks (liquors) retard digestion.

Tea and Coffee.—Coffee stimulates the secretion of the gastric glands and increases the peristaltic movements of the intestine. Tea has a constipating effect on account of the large amount of tannic acid it contains, and in animal experiments it retards the secretion of acid and delays the peptonization of protein substances. Coffee should be forbidden in most cases of gastric disease. Very weak tea, on the contrary, may be taken with advantage, especially if used as a vehicle for milk or other nutritive materials. In health, however, there is no reason for apprehending danger to the race at large from coffee-drinking. Coffee-drinking has not affected Americans to any appreciable degree, though coffee has been the almost universal beverage for many decades. The life insurance companies, constantly warring against everything that tends to shorten life, are silent in regard to coffee as a beverage. The experiments of Chase on three normal individuals who were not addicted to tea or coffee show that when taken with meals, in the amounts ordinarily used, these beverages do not retard either salivary or peptic digestion. It has been found that salivary digestion is aided slightly by tea. Both tea and coffee may act as mild stimulants to gastric secretion; the digestive power of the secretions, however, is not augmented, but, on the other hand, neither is it impaired, as in the use of whisky. Therefore, as a stimulant to gastric secretion, tea or coffee would seem preferable to whisky. A great deal has been said about the deleterious effects of tea and coffee on the stomach. Apart from their stimulating effect on the central nervous system, if properly made and not too strong, their effect on digestion is almost neutral.

Cocoa.—Cocoa possesses much higher nutritive value than tea or coffee. It does not stimulate gastric digestion, and prepared with either water or milk it is a proper beverage for patients with stomach disease. Preparations of cocoa from which the oil has been expressed, and which have not been treated with alkalis, are to be recommended. Chocolate prepared by admixture of sugar and spices is not so easily digested. It contains a larger proportion of fat and carbohydrates, and may therefore give rise to fermentation and the formation of acid.

Tobacco.—The use of tobacco in any form is to be interdicted in all cases of stomach disease because, clinically, it has often proved to be the cause of chronic gastritis and its sequelae. Nicotin may reduce the peristaltic motions of the stomach in consequence of its paralyzing effect on the vagus. Tobacco may cause hyperacidity in the empty stomach. Smoking after meals induces salivation, and when the saliva is swallowed the acid secretion of the stomach becomes neutralized.

Instructing the Patient.—The patient must be definitely instructed in regard to what articles of diet are permissible and what are not. Printed schedules are frequently provided for this purpose. The

allowable and the forbidden foods are all enumerated on these, and those unsuitable for the patient are crossed off. The physician should aim at avoiding unnecessary monotony in dietary arrangements. If possible, food luxuries and spices should be permitted to such an extent as to render the prescribed diet relishable.

It is quite proper, indeed necessary, to give exact counsel regarding the quantities of food to be taken; and the physician should not confine himself to such general measurements as spoonfuls, cups, and wine glasses, the standards of which vary so widely. The quantity is more accurately specified in grams, as: Of fruit preserves, the portion for a patient with gastric disease should not exceed 150 grams (five ounces). Hints regarding the mode of preparation of food must likewise be carefully given—e. g., whether meat should be eaten raw, boiled, or roasted; in what form and state of subdivision the various foods are to be taken (purée, mashed, etc.); to what extent fats and spices may be employed in the preparation of the dishes; and how strong tea or coffee may be made.

It is quite essential, too, to impress upon the patient the number of meals to be taken, and at what hours. The rule is, light meals at frequent intervals. This holds good particularly in cases of atony, dilatation, and pyloric stenosis, because in such conditions large quantities are very difficult to manage. In some cases where hypersecretion is a feature the intervals between meals should be extended in order to provide, if possible, adequate periods of rest for the irritated gastric mucous membrane. Deviations from the usual dining schedule should be as infrequent as possible. Irregularity in eating is apt to prolong the stay of the food in the stomach. The patients should not retire earlier than two hours after partaking of the evening meal. Mastication and oral digestion are of the utmost importance. Only when these are accomplished in a correct manner is it possible for the food to reach the stomach in such a condition as to facilitate its penetration and solution by the digestive juices.

Patients should eat slowly. Prolonged mastication not only thoroughly insalivates the food, but has a favorable and stimulating effect on the secretion of the gastric juice. During the meal no strain should be put upon the mind, consequently reading while eating is to be forbidden. Anger, excitement, and irritating discussions must be avoided at the table. When the patient has no appetite he is not to be coaxed or harassed into taking food.

Beverages in moderate quantities are, as a rule, without evil influence in health. In gastric diseases, however, drinking during the meals is probably better omitted.

Immediately after eating, fatiguing bodily or mental exercise should not be taken. Vigorous bodily exertions at such a time produce, even in health, sensations of discomfort; in disease they

are positively harmful, as they are liable to diminish the secretion of hydrochloric acid in the stomach. A patient with gastric disease should lie down after eating, and on his right side, since in that position the stomach is emptied more rapidly. The clothing should not bind the stomach.

It is an open question whether patients should sleep after dinner; in the majority of cases this may be left to the patient himself. The percentage of acid in the stomach, and the motility of that organ, are said to be diminished during sleep. These facts must be borne in mind when considering the advisability of either forbidding or permitting the after-dinner nap.

CHAPTER VII.

DIET IN INTESTINAL DISEASES.

REGULATION of the diet plays the chief rôle in all therapeutic measures employed in the treatment of intestinal diseases. Broadly speaking, the various affections of the bowels are characterized by either diarrhea or constipation. For this reason the dietary instructions must, as a rule, be formulated with special regard to the constipating or laxative effect of the selected foods.

CONSTIPATING DIET.

A constipating diet is one which will not stimulate peristalsis of the intestine, either mechanically or chemically; it does not irritate the mucous membrane, muscles or nerves of the intestine. Food that is imperfectly acted upon by the secretions of the stomach and intestine, resulting in fermentation and putrefaction, should be interdicted, since the products of fermentation and putrefaction are great stimulants of peristalsis. The patient must be instructed to eat sparingly, so as to limit the elimination of bulky or waste products which excite normal peristalsis. Foods should be taken which, on absorption, leave little or no residue of which the intestine has to rid itself. Astringent foods and beverages inhibit both normal and abnormal secretion, inducing constipation. Small and frequent meals prevent overdistention and retard peristalsis.

A constipating diet, which in effect is equivalent to a non-irritating diet, must be considered in the following conditions if they are associated with diarrhea, viz.: Acute and chronic catarrh of the small and the large intestine, chronic diarrhea in its various forms, hemorrhages from the bowel, intestinal ulcers, malignant neoplasms, and stenoses. The time has passed when every case of diarrhea could be classed as "intestinal catarrh." It is of the utmost importance that an exact diagnosis be established before dietetic measures are instituted. Today we must in every case determine exactly the work done by the intestine, by examining the feces. In the opinion of the author the method of determining the physiologic activity of the intestine, inaugurated by Schmidt and Strasburger,¹ and culminating in the well-known test diet, deserves to be preferred to all other methods. It is a fact that all the attempts—and there have been many of them—to modify the practice of

¹ Schmidt and Strasburger: *Die Fäzes des Menschen*, Berlin, 1910.

Schmidt and Strasburger have been unsuccessful; they do not represent any improvement in method. Macroscopic, microscopic and chemical examination of the feces after Schmidt's test diet¹ must be followed out in its entirety before the rules for a rational dietary regimen can be laid down. (See Chapter IV.)

By means of examination of the feces we are frequently able to locate the pathologic process in the large or the small intestine—a very important differentiation in its bearing on dietary restrictions. We can also by this means obtain information as to whether some particular constituent of the food (meat, carbohydrate, fat, connective tissue) is exceptionally ill-digested. Moreover, examination of the material of a single evacuation will show whether protein putrefaction or carbohydrate fermentation is present. The knowledge afforded by analysis of the feces has to be carefully borne in mind in planning the patient's regimen, for upon it depends the question of increasing or decreasing the amount of protein, carbohydrates or fats to be taken in any particular case, or the adoption of a different and easily digested diet. It is particularly important to establish positively whether there is putrefaction or fermentation. It is possible, by making a change in the diet in these cases, to create a nutritional medium in which the harmful or undesirable bacteria will be unable to multiply with their usual rapidity. The examination of the bacterial flora of the intestinal tract with this end in view is not merely of theoretical, but of practical importance. In the large majority of cases the treatment to be sought is the one which most effectively combats the putrefactive processes within the intestine.

It is now known that the fluidity of diarrheal discharges is due to the transudation of serum into the intestinal canal. This serum, as well as other products derived from the intestinal wall (mucus, pus, blood), is particularly liable to decomposition; it "putrefies" much more readily than protein of the food which may have escaped digestion. For this reason, in all chronic diseases of the intestine (catarrhs, ulcers, ulcerating malignant tumors) there is found a decomposing stool, which may be recognized as such by analysis of the test-diet feces (decomposition in the incubator test, with a change to an alkaline reaction, and occasionally, though more rarely, the demonstration of soluble protein). Cases of putrefaction are to be differentiated from those of pure carbohydrate fermentation—that is, from the cases of chronic intestinal disease in which fermentation of carbohydrates predominates (recognizable by acid fermentation in the incubator test). These latter conditions are comparatively rare. (See page 116.)

Gastrogenic diarrheas, especially when existing for a long time, are quite often associated with catarrhal conditions of the intes-

¹ Adolf Schmidt: *The Test Diet in Intestinal Diseases*, translated by Charles D. Aaron. Philadelphia, 1909.

tinal mucous membrane, and in these cases the intestinal contents incline more toward putrefaction. (See Chapter XXXVIII.)

From these facts we must conclude that in the majority of intestinal diseases accompanied by diarrhea, that is to say in the overwhelming majority of all patients suffering from intestinal ailments, we must insist upon a diet opposed to putrefaction.

A diet may act antiseptically because of either its chemical or its mechanical constitution. From the chemical viewpoint, in an antiseptic diet the carbohydrates must predominate. We should either eliminate entirely protein articles of diet, especially at first and in grave cases, or restrict them to those protein substances which are most easily soluble and most readily digested. Where both carbohydrates and protein bodies are in the same patient subjected to decomposition, the putrefaction of the protein is retarded as long as there are present carbohydrates capable of fermentation. From the mechanical standpoint an entirely non-irritating diet must be demanded. It is evident that a diet that irritates the bowel as little as possible will diminish the secretion of easily decomposable serum, mucus, and pus, and will thus contribute to the diminution of the processes of decomposition. This non-irritating intestinal diet is obtained by the most minute subdivision of all articles of diet, the removal of all coarse residues (cellulose, connective tissue) from the food, and the avoidance of all additions of irritating spices. It is by no means an easy matter to arrange a menu conforming to all these requirements and at the same time giving taste, flavor, and variety. The best antiseptic food, both chemically and mechanically considered, is afforded by the mucoid soups—which should therefore be employed in all grave cases. The mucoid soups are prepared from oatmeal, rice meal, wheat starch, rice starch, potato starch, and corn starch. The grains, grits or flakes are boiled four to six hours with water, passed through a fine sieve, and again brought to the boiling-point—when they are ready for use. The flours, beaten up in a small quantity ($\frac{1}{2}$ liter—4 ounces) of cold water, are added to $\frac{1}{2}$ liter (1 pint) of boiling water and boiled four to six hours. By proper additions of milk, cream and butter these soups are rendered palatable and nutritious. In cases of lesser severity, soups may be given prepared from other leguminous flours, from tapioca, sago, ground rice, dried rolls, or zwieback and cocoa.

Antiputrefactive Diet.—Of the foods containing protein and acting antiseptically, milk deserves particular attention. A constant endeavor should be made to accustom the patient to it. Milk, though it possesses great nutritional value, requires for its assimilation comparatively little digestive work. It has also been ascertained that milk, of all the foods containing protein, yields the smallest proportion of decomposition products.

Among the medical profession as well as the laity, milk is frequently looked upon with disfavor as an article of diet in cases of gastric disease, under the mistaken impression that it is difficult of digestion. It is true that milk occasionally causes diarrhea and sometimes constipation, both in health and in those suffering from intestinal disorders. This is, strange to say, the case when the milk is taken pure (fresh or boiled) and without admixture. These undesirable effects may be avoided by adding to the milk minute quantities of rice, oatmeal, crackers, or other food; in the course of a few days after this change is instituted the milk will, on ingestion, be assimilated without difficulty. Schmidt is of the opinion that in cases in which the patient has not been drinking any milk for a long time, the intestine and its vegetation may need a certain time to adapt themselves to the new food. As yet no satisfactory explanation has been advanced for those rare cases in which milk always gives rise to diarrhea by its peculiar influence on the peristaltic movements. The cause cannot be found in the ingredients of the milk. Milk is the mildest and least irritating of all foods, being digestible even by the tender digestive organs of the newborn infant. When milk is not well borne, it may be assumed that, because of certain individual peculiarities or pathologic conditions, it is easily decomposed, and that the intestine is irritated by such decomposition products; or the intestine, being in a state of hyperirritability, is abnormally sensitive toward the decomposition products of milk digestion. It is possible that this peculiar irritating effect of the milk may be brought about by a rapid fermentation of the carbohydrates (milk-sugar). Of all the varieties of sugar, milk-sugar most readily yields to fermentation within the bowel. It must, however, be distinctly understood that these disturbances after the ingestion of milk, though undoubtedly occurring in isolated cases, are by no means of so great frequency as is generally supposed, even in patients with intestinal disease. Credit should not be given unhesitatingly to the statements of certain patients that they are unable to digest milk, for on closer investigation it is usually found that the milk is perfectly assimilated. For these reasons Schmidt has made milk an integral constituent of the test diet he recommends (*vide infra*).

No cause, therefore, appears for abandoning milk in the feeding of patients suffering from intestinal disease; the more so since it is no easy matter to replace this excellent and easily digestible food by other substances. The possibility, however, that disturbances may occasionally take place in the digestion of this all but ideal food, should induce us to exercise great care and circumspection in the administration of a milk diet. The best guide is the degree of digestibility of the milk administered during the test diet for analytic purposes. Our practice, therefore, consists at first in the giving of small quantities of milk as an addition to other foods. Such foods are acorn-cocoa, cocoa, chocolate, and various muc-

luginous foods prepared from rice, tapioca, barley, oats, or different kinds of flour. Milk may also be employed generously as a constituent of the numerous kinds of mucilaginous and flour soups, mushes, puddings, and purées. With the exercise of care in the administration of the milk, beginning with $\frac{1}{2}$ liter ($\frac{1}{2}$ pint) per day, the quantity may be rapidly increased to $\frac{3}{4}$ liter (1 pint) and even more. Should increased peristaltic movements be noticeable during the first two or three days (the occasional constipating effect of milk would be rather welcome in the cases under discussion), this should not be regarded as a reason for discontinuing the milk, for tolerance usually becomes established in a few days. If it should be necessary to discontinue the milk, as may occasionally be the case, it is advisable to insist upon repeated trials as to its toleration. Milk should be used most carefully in all cases of well-marked catarrh of the small intestine and in cases of ulceration (tuberculosis), for in these diseases the irritating effects are most frequent; but it may be given in larger quantities when the disease is located lower down in the bowel. In certain chronic catarrhs of the large intestine, milk frequently has a very beneficial effect.

Adolf Schmidt has drawn attention to another means employed for diminishing the irritating effects of milk. He reasoned, from the belief that the irritating effects of milk in many cases are due to its abnormal facility of becoming decomposed, or to the abnormally increased sensitiveness of the intestinal mucous membrane toward the decomposition products of the milk, that the addition of salicylic acid to the milk, by counteracting the tendency to decomposition, would render the milk tolerable. For this purpose he proceeded as follows: The daily portion of the milk intended for consumption was boiled with 0.2 Gm. (3 grains) of pure salicylic acid per liter (quart). First the proper quantity of salicylic acid was triturated with a little milk in a mortar, and only when minutely subdivided was it added to the total quantity (1 liter). It has been shown that milk prepared in this manner is well borne in cases in which chronic hypersensitiveness toward pure milk is present, not only in adults, but also in children. The addition of salicylic acid removes the deleterious qualities of the milk without interfering with its good qualities. For the same purpose we frequently add one or two tablespoonfuls of lime-water to a glass of milk. Salicylic acid, however, is certainly more effective.

In the feeding of young patients the milk should be administered only after being boiled, in order to prevent the accidental introduction of pathogenic germs (typhoid, tuberculosis, diphtheria, scarlet fever). This precaution does not appear to be necessary, however, in the case of adults.

Milk may be replaced by any of the preparations of milk fully described in Chapter VI—pegnin milk, milk with the addition of cream and milk-sugar, buttermilk, etc. Special attention should

be given to three-day kefir, which acts as an astringent, and particularly to yoghurt, the disinfecting effect of which, together with its great nutritional importance, has already been fully considered. (See page 162.)

Should it be desirable to administer other protein substances besides milk, and in severe cases it is sometimes not only desirable but necessary, a protein should be selected that is non-irritating and antibacterial. Among the artificial food preparations, those containing casein in small quantities (nutrose, plasmon) are deserving of special consideration. It is a question whether in severe cases gelatin should not be used more extensively than it is today. Gelatin belongs to the nitrogenous group of substances; it possesses many of the aminoacids in common with protein, and is easily converted into peptones by the digestive ferments. Gelatin does not contain either the tryptophan or the tyrosin radicals that are always found in protein, and for this reason does not readily decompose. Tryptophan in the process of decomposition forms skatol, indol and other irritating products, while tyrosin evolves several varieties of phenol, which in case of excessive putrefaction may be absorbed, producing toxic symptoms. Gelatin is easily digested, and readily absorbed by the intestinal mucous membrane.

Recently we have learned the value of gelatin in certain gastro-intestinal diseases. Murlin found that under certain conditions as much as 43 per cent. of the total nitrogen necessary for the body could be supplied in the form of gelatin. Herter considers gelatin a valuable food, possessing the property of inhibiting certain forms of intestinal decomposition. He calculates that one ounce of gelatin will yield 120 calories. This amount may be easily added to milk and may thus at times be of the greatest importance for nutrition. It has been found that certain microorganisms (*Staphylococcus aureus* and *Bacillus infantilis*), commonly found in the intestinal canal in certain gastro-intestinal diseases, will not grow on a gelatin medium outside of the body, while on a carbohydrate or protein medium they multiply rapidly. This is a logical reason why gelatin should be used more than it is in intestinal diseases. (See page 161.)

The value of this non-irritating diet is strengthened by the fact that the volume of the food is small. This circumstance permits as great an increase in the percentage of calories contained in the food as it is possible to furnish—a result that is attained by the addition of cream and the best kind of butter. Additions of beef bouillon, meat extract, maggi, and common salt, in moderate quantities, are desirable and even imperative, not only for their flavor, but also for their stimulating effect on the appetite. Besides this the astringent effect of some articles of food should be utilized, as for example tea, worm-curd, claret rich in tannin (especially the Greek camerite), and particularly whortleberries. The latter are given in the form

of decoction, whortleberry juice, whortleberry thick soups, whortleberry wine, and whortleberry gelatin. The skins and the seeds must be entirely removed. Blackberries, also rich in tannin, may be employed in a similar manner.

Such a diet will always assist in accomplishing the desired end, which is to inhibit or to diminish well-marked processes of decomposition. It is possible to diminish the number of bacteria in the feces by the use of antibacterial soups, milk diet, and salicylic milk. Under this regimen, should pure carbohydrate fermentation develop, which could be ascertained by an analysis of the feces, no harm would be done. It would be preferable to the putrefaction of protein, and could be promptly inhibited by limiting the supply of carbohydrates or by the use of salicylic milk. When in severe cases improvement begins, other articles of food are added gradually. It must, however, be stated that extreme caution should be observed, and the addition of more food should take place in the most gradual manner. All food must be of the aseptic variety and free from all irritating properties; otherwise relapses are apt to occur. The basis of the diet will therefore consist of soups and milk, the quantity of which may be gradually increased and a somewhat greater variety permitted. In this respect foods containing carbohydrates are to be considered first, the additions consisting of zwieback, crackers, and stale white bread, all of which must be well softened and thoroughly masticated. The soups are thickened with thoroughly cooked rice or browned flour. The quantity of bouillon, and especially the quantity of butter, may be increased—the latter, of course, only in those cases in which the feces do not contain abnormally large quantities of fat. Later, soups may be gradually omitted; they may be replaced by various kinds of mushy foods prepared from rice, sago, oat flakes, tapioca, various flours with the addition of butter, milk, cream, whipped cream, claret, or whortleberry wine with sugar. Frequently the addition of sugar must be restricted, and occasionally saccharin may be used. As the case progresses, light flour dishes or noodles should be given. Gradually and carefully vegetables may be added; these, however, must be well ground up and forced through a sieve—*e. g.*, small quantities of mashed potatoes prepared with plenty of butter and eggs; purée of white beans, peas, cauliflower, or artichokes, with the addition of butter, bouillon, and milk. When the compact form of the feces indicates that transudation of serum through the mucous membrane has ceased, and when mucus is being excreted in small quantities only, and the incubator test shows that there is no decomposition in the feces, then no fear need be entertained that putrefactive processes will be reestablished by the careful resumption of proteins. Jellies, being easy of digestion, should be allowed first—meat jelly, milk jelly, whortleberry jelly. Then the artificial albumino-casein preparations may be

given in larger quantities as adjuncts to soups, etc. When these are well tolerated, eggs can be added. Eggs may be taken in a great variety of preparations, as yolk of eggs in beef-tea and soups, the whole egg in omelets or light puddings, as a component of jellies and pies, or beaten up to a froth with claret and a little sugar added. Caution, however, is always necessary with eggs, since patients suffering from intestinal disease are apt to be singularly intolerant of them. Later, small quantities of tender meat may be allowed (squab, breast of chicken, and tender lean beef), all of which must be well cut up and chopped or forced through the very finest of hair sieves. The meat may either be given as such or stirred into soups. Soups prepared with calf's brain (demenbrated) and veal sweetbread may be given. Schmidt demands, and justly so, the exclusion of raw, smoked or pickled meat from the diet of patients suffering from intestinal diseases, not only because of the toughness of the material, but particularly because it may contain somewhat coarse shreds of connective tissue. This is particularly important in cases of gastrogenic diarrhea, for the raw or smoked connective tissue is not dissolved by an achylie or subacid and muscularly weak stomach. It has been found that raw connective tissue is not always dissolved by normal or even hyperacid stomachs. Reaching the intestine in an undigested condition, the connective tissue is not dissolved at all, but constitutes a coarse mechanical irritant to the intestinal mucous membrane, especially if the latter is diseased. Besides, it becomes a breeding place for a vast number of microorganisms of putrefaction and fermentation. Raw beef is only to be given when scraped carefully with the spoon from the whole cut of a tender fillet; in such scraped meat the amount of connective tissue is reduced to the smallest possible proportion.

At this period the diet should approximate the test diet of Schmidt (page 112). To this diet when necessary some claret, a little bouillon and at night thin slices of cold roast veal may be added.

The Schmidt diet is an example of a diet non-irritating to the intestine, and as such valuable not only diagnostically but therapeutically. It may in many cases be made the basis of the general diet, to be varied as occasion demands by increasing or diminishing the different articles.

In connection with this diet green vegetables are permitted in small quantities when the cellulose they contain does not act as an irritant. Only those which may be easily cut up into very fine particles are allowed (spinach, cauliflower, green lettuce, ends of asparagus). As the improvement continues and the last-mentioned foods are well tolerated, the consumption of larger quantities of all these foods is gradually permissible, and later the coarser ones need not be cut up as minutely as before. Then boiled fruit is allowed, at first in the form of jam (apples, pears). The skins and

seeds must be forbidden for a long time, also those varieties of fruit which contain much acid, since the fruit acids may act as purgatives. The object of this gradual change in the character of the food is the rehabilitation of the intestine to the usual mixed diet as tolerated without any discomfort in health. This desideratum is, unfortunately, not always attainable in chronic cases, especially in chronic catarrhs, which are often very obstinate. In such cases the diet should be kept under control and should consist of non-irritating foods for the remainder of the life of the patient.

Regarding beverages, warmth of the liquid must be especially advised in grave cases in which the diet has been carefully arranged. Warm drinks have a calmative effect on the mucous membrane of the intestine. Various kinds of teas are given, as chamomile, anise-seed, valerian, and peppermint, with or without the addition of milk, claret, rum, or cognac. Besides these, there may be given boiled water with claret, pure milk, claret, and whortleberry wine. Lemonades and fruit juices are only to be given during convalescence. Ice-cold drinks are to be positively interdicted.

Thus far the dietetic treatment of severe cases with diarrhea, marked decomposition, and excessive peristalsis, has been described. It is evident that for moderate and light cases the diet from the start need not be arranged quite so carefully. The light cases commence with a diet which is equivalent to the one permitted in severe cases during convalescence. The guide here also may be the tolerance of the intestine toward Schmidt's test diet. In light cases the test diet may be given from the beginning for therapeutic purposes.

It is therefore essential to individualize the nutrition in the cases of intestinal disease under discussion, basing both it and the diagnosis on the results of the analysis of the feces. Should the stomach be simultaneously affected, its digestive ability must have attention, since in many cases of chronic diarrhea (gastrogenic diarrhea) the treatment of the stomach is of greater importance than the most careful intestinal diet. The same holds good in cases of nervous diarrhea. The treatment here has to be directed particularly toward the nervous system. A strict outline of diet cannot be propounded for nervous cases. Adherence to a non-irritating diet is often unnecessary in these cases, and may even prove to be harmful. A sudden change in the nervous cases frequently produces a surprisingly beneficial effect. Generally speaking, the dictum holds good that we should be more careful in the diet the higher up in the intestine the disease is situated. The greatest care is demanded in diseases of the small intestine; when the pathologic process is located in the lower portion of the large intestine it is not necessary to be nearly so strict. The reason for this is that the contents of the small intestine normally enter

the large intestine in a semiliquid state; if, therefore, the small intestine functions properly, both it and the large intestine are protected to a certain extent from mechanical irritation by the intestinal contents; but if the function of the small intestine is defective, irritation follows, not only to this part of the alimentary tract, but to the large intestine as well. In diseases of the large intestine, every kind of food should be avoided that cannot be easily transformed into a semiliquid mass in the small intestine (connective tissue, cellulose), but there is no necessity of being so particular about the minute breaking up of the food as in diseases of the small intestine.

Antifermentative Diet.—The antifermentative diet stands in contrast to the antiputrefactive. It is indicated in all cases in which fermentation of the carbohydrates causes acute or, particularly, chronic diarrhea. Such carbohydrate fermentation is found occasionally in all chronic diseases of the intestine, and especially in acute and chronic catarrhs and in gastrogenic diarrhea when the bacteria of fermentation succeed in settling in the intestine on a favorable soil containing carbohydrates. The organisms responsible for the fermentation of the carbohydrates are the iodine germs, so named by Schmidt. They contain granules, and become blue in the presence of iodine; unstained they resemble yeast cells. They are probably identical with the *granulobacillus butyricus* of Gräbner. Thus there are intestinal catarrhs and gastrogenic (hyperacidity) intestinal disorders in which the diarrhea is not associated with putrefaction, but with fermentation. These latter cases, however, are rarer than those in which decomposition of protein predominates.

A disease of this kind, of rather frequent occurrence, is the so-called intestinal fermentative dyspepsia of Schmidt and Strasburger, which may appear in the absence of any organic lesion of the intestine and when the stomach is perfectly normal (see Chapter XXXVIII). Fermentative changes are recognized by the light greenish-yellow color, the spongy semifluid consistency, and the strongly acid reaction of the feces. In the incubator such feces always show an acid fermentation (Plate VIII, Fig. *b*). These cases demand a careful regulation of the diet, as determined by analysis of the feces. One can also learn by this analysis whether the bowel is organically diseased, or whether the condition is due to a pathologic stomach. The principle of the diet naturally consists in reducing the amount of fermentable carbohydrates, or discontinuing them entirely. Protein and fat should be substituted and continued until examination of the feces shows that the ferments have lost their activity. When the intestine is entirely healthy no special attention need be given the gross form of the diet; it is only necessary to supply a pure protein-fat diet for a certain length of time and then gradually administer the most easily soluble or even

predigested carbohydrates until tolerance is reestablished. But when there are synchronous organic intestinal disturbances, particularly catarrhs, the diet has to fulfil all the above stated requirements respecting the absence of irritants and the minute subdivision of the food, in proportion to the gravity of the pathologic condition. Here, too, it will be impossible to give protein and fat exclusively, because the fermentation process is readily convertible into an undesirable decomposition of protein; still the proportion of carbohydrates must be reduced. The diet should at first consist of watery soups with the addition of beef-tea, butter, artificial albumins, Maggi, and a small quantity of dry rolls or zwieback. After these have been shown to be well borne, oat or wheat flour, dextrinized infant foods or flours, zwieback, toast, hominy, noodles and rice should be given in small quantities, with the addition of protein nutrients such as eggs, milk, bouillon, very tender chopped meats, chicken, squab, and fats in the form of butter and oil. The quantity of the diet should then be gradually increased until solid food is allowed, the major part consisting of proteins and fats, the use of carbohydrates being restricted. By following out a diet of this kind it will usually be possible to rapidly reduce the fermentation processes. Potatoes should be forbidden for a long time, as they are poorly dextrinated in the intestines of such patients, and in consequence the starch they contain undergoes fermentation. Care must likewise be exercised in respect to milk when fermentation is present. Milk-sugar ferments very easily; it is therefore advisable to add salicylic acid when milk is given.

When the stomach is the primary cause of the intestinal disorder, it naturally must first have proper treatment.

LAXATIVE DIET.

A laxative diet is one which increases the peristalsis of the intestine, chiefly by stimulating its muscular coat. This effect is brought about by the mere bulk of the food or by its chemical properties. In all conditions of chronic constipation it is necessary to examine the feces most carefully in order to be able to outline a correct dietetic course. The causes of constipation are so various that an exact analysis of the feces after a test diet is essential to a correct understanding of the case. The diet will have to be different in cases of pure atonic constipation from that which is appropriate in spastic constipation. Again, it will differ when the constipation is due merely to weakness of the abdominal muscles or of the rectum.

A laxative diet is the exact reverse of a constipating or non-irritating diet. In its use both mechanical and chemical stimuli are employed; the food is coarse and plentiful, and deliberately calculated to increase the processes of decomposition and fermentation.

The most powerful dietetic stimuli are necessary in cases of atonic constipation in which the bowel is relaxed and its peristaltic motility lost. Stimulation of a decidedly weaker order, chiefly of a chemical nature, is employed in those varieties of constipation which are described as spastic, in which the intestine is frequently overstimulated. Chemical stimulation is also useful in those forms of nervous constipation which are associated with intestinal catarrh and abnormal secretion of mucus.

By making the diet large and bulky the mechanical effects are obtained. By this means it is possible to increase the quantity of the fecal excrement, which is usually very small in chronic constipation. This object is attained by the giving of a diet rich in insoluble residue, usually an increased amount of carbohydrates, and more particularly of foods rich in cellulose. Quite erroneous views were formerly entertained, and even survive at present, with regard to the behavior of cellulose in the human intestinal tract. It has always been assumed that the human organism does not possess a digestive ferment for cellulose, and that the latter is not subject to changes by the digestive juices in the lower sections of the intestine. It has also been supposed that, particularly in the large intestine, cellulose undergoes a sort of fermentation, due to the influence of bacterial activity, being converted thereby into marsh gas, hydrogen, acetic and butyric acids. Lohrsch, however, within the last few years has demonstrated, as the result of numerous experiments on assimilation and respiration, that cellulose in the small intestine behaves in exactly the same manner as the ordinary carbohydrates. Just like starch, it is converted into sugar and absorbed. This necessitates the existence of a cellulose ferment—a cytase. The transformation of cellulose into sugar proceeds much more slowly than that of starch, for the reason that cellulose by its very nature is a hard, brittle material, and because in plants it is present, not in the pure state, but mixed with and enveloped in the so-called encrusting substances (lignin, cutin). This makes it very difficult for the digestive juices, in particular the cytase, to dissolve the cellulose. The digestion of cellulose is more readily accomplished if the plants from which it is derived are quite young, because then they contain less encrusting substances and are more easily subdivided. (See page 61.)

According to Lohrsch, cellulose is digested when introduced with a test diet in the following proportions: Average, 58 per cent.; tender cauliflower, 79 per cent.; mashed spinach, 90 per cent.; bread-cellulose, 85 per cent.; and the entire quantity of tender white cabbage may be digested under certain circumstances. It may therefore be considered as a settled fact that in man nearly all of the cellulose is regularly digested, only a small quantity being expelled with the feces. A very small fraction of undigested cellulose may have to undergo fermentation in the large intestine.

Cellulose, naturally, will never attain much importance as a food, the quantity contained in plants being so small.

It follows from what has been said that if it is intended by means of a diet rich in cellulose to exert a laxative effect upon the intestine, the food must be as coarse as possible, comminuted only slightly, and not too tender or too young. Otherwise the intended effect of the feeding will be a disappointment because of the readiness with which the small intestine digests the cellulose. This is, in fact, what frequently happens in cases of chronic atonic constipation. It is true that in a large number of cases of chronic constipation a good effect is obtained by a diet rich in indigestible residue, but in many of these cases the ingestion of large quantities of cellulose is without any effect, and it has been found that these patients digest cellulose a great deal better than people with normal intestines. Lohrlich was able to demonstrate that persons with habitual constipation, fed in the same manner as normal persons, would digest about 80 per cent. of the cellulose introduced into the intestine, in contradistinction to the normal standard of only 58 per cent. This peculiar fact explains the negative effect of cellulose in many cases of chronic atonic constipation. Such individuals are capable indeed of digesting everything, even the coarsest gritty foods (nuts, almonds), without leaving sufficient residue to be of any importance for stimulating peristalsis.

However, in every case of chronic atonic constipation the attempt should be made to provide a diet rich in coarse residue, for it is always a matter of uncertainty how the intestine will behave in respect to cellulose. Various kinds of bread should, therefore, be used unsparingly, *e. g.*, rye bread, pumpernickel, Graham bread, corn bread, ginger-cake and honey-cake. Other special varieties of bread are Rademann's cellulose bread (3 per cent. cellulose) and Weicker's cellulose bread made of beechwood sawdust. Of vegetable foods, various kinds of lettuce and coarse vegetables, such as cabbage, carrots, turnips and parsnips, should be freely utilized.

Large quantities of raw and cooked fruit are always valuable; to a certain extent they answer the indication of making the feces rich in water and consequently softer. To get nearer to this desideratum Adolf Schmidt has proposed that we give agar. This agar is a well-known species of Japanese sea-alga and belongs to the so-called hemicelluloses, being anhydrides of various kinds of sugar (except dextrose). These hemicelluloses are, chemically, polysaccharids, closely related to cellulose. Agar is the anhydrid of galactose; it therefore consists of galactan, and on being hydrated yields about 70 per cent. of galactose. This hemicellulose, as Lohrlich has shown, is digested in the human organism very much like cellulose, but in much larger amounts, and it can be administered in considerably larger quantities. A constipated person will digest much more of it than a normal

person. Still, if large quantities are given, enough may remain undigested to actually soften the stools to a semiliquid consistency (see page 61). Large amounts of fat are capable of acting in a similar manner, rendering the feces soft and smooth. Benedict was the first to draw our attention to a similar effect from liquid petrolatum taken internally (see page 664). These substances may therefore be employed advantageously to increase the effects of a diet rich in coarse residue.

An immense variety of fruits must be classed among the laxatives, because of their chemical constitution. These are of particular value in cases of spastic constipation. They stimulate peristalsis partly because of their fruit acids and partly because they contain sugar, which is apt to increase the fermentative processes in the intestine. Fruit and its active ingredients may be freely used as jams, jellies, fruit juices, cider, etc. Other acids and acid foods are recommended, as citric acid, lactic acid, buttermilk, sour milk, whey, kefir (two days old), sour cream, yoghurt, vinegar and sauerkraut (See page 654.)

The most useful kinds of the various sugars are the easily broken-up milk-sugar and levulose. Easily melted fats, as butter, oil, and cream, not only have a mechanical effect, as has been mentioned, but also act chemically, stimulating peristalsis by means of the great amount of fatty acids they develop. Some beverages have a certain chemical action, as acidulous beers (Weissbier) and beverages containing carbon dioxide.

All these articles of diet frequently act particularly well when taken on the fasting stomach. Their temperature is also of importance. When taken cold, the stimulation of peristaltic motion is often much enhanced. This susceptibility of the intestine to cold goes far to explain the effect of a very cold glass of water taken in the morning immediately after rising.

No specific directions, as a rule, need be given concerning the consumption of meat in cases of constipation. Meat may be taken freely in spastic constipation, though some authors recommend a restriction or even a prohibition of it, as it is said that it occasionally induces constipation.

The diet in the different varieties of constipation, generally speaking, is to be formulated in such a manner that a mechanical and chemical stimulation will be produced. An active diet rich in insoluble residue is particularly indicated in chronic atonic constipation. In such cases there need be no misgivings in advising large quantities of coarse food so long as the stomach is in normal condition. Should the stomach be deranged, the ingestion of cellulose will have to be considerably moderated, and attempts should be made to proceed on other lines. Here, also, it is imperative to treat every case individually. In cases of spastic constipation the indications point to a chemically acting dietetic regimen

exclusively; all kinds of mechanically irritating foods are to be absolutely forbidden. In many cases it may be necessary occasionally to administer vegetables and boiled fruit in purée form, our only dependence being the mildly laxative chemical action of appropriate foods.

All the articles enumerated previously as producing a constipating effect must be absolutely avoided (claret, tea, cocoa, acorn-cocoa, whortleberries).

CHAPTER VIII.

ARTIFICIAL FOOD PREPARATIONS.

IN cases where the general nutrition is low and only small quantities of food can be ingested, it has been found necessary to supplement the "natural" diet by the use of specially prepared nourishing agents. We have a large number of such preparations at our disposal.

To take the place of proteolysis in the stomach, which is so frequently deficient in chronic gastro-intestinal diseases, a number of nutritious preparations are manufactured in which the protein is predigested into peptones and albumoses. Preparations of this class are not necessary when the patient is able to digest sufficient food for his requirements; but they are indicated in cases where the general nutrition is low. Many of them are, however, impracticable, owing to their disagreeable taste; and the cost of those that can be used is generally so high as to curtail their usefulness among patients in moderate circumstances.

The protein preparations are made by artificial digestion of protein by means of animal and vegetable ferments with the aid of organic and inorganic acids, salts, bases, vapors, and gases, in a vacuum or under high pressure. The principal preparations of this class include the following:

Preparations of Animal Protein.—*Somatose* is a yellowish powder, nearly tasteless and odorless, and readily soluble in water. It contains over 90 per cent. of albumoses, is easily assimilated, and stimulates appetite and gastric secretion. It has been employed with benefit in chronic gastritis, in gastric crises after surgical operations on the stomach and intestine, in carcinoma, in nervous dyspepsia and anorexia, and in acute gastroenteritis. *Somatose* is, however, not well borne in hyperacidity. The dose is three or four dessertspoonfuls a day. Its proper use is as an adjuvant in connection with the prescribed diet, to supplement the nutritive value of the latter. *Somatose* has the action of a tonic rather than that of a food. Iron *somatose* has been furnished by pharmaceutical houses. In iron *somatose* the iron is organically combined; this preparation is indicated in cases of chlorosis complicated with gastro-intestinal disturbance; the adult dose is three or four dessertspoonfuls daily.

Carringen, or *somatine*, occupies a place between somatose and meat extract as regards composition; its effect is stimulating. Its cost renders it impracticable as a food.

Tropon is prepared from animal and vegetable protein, and is useful as a cheap meat powder. It contains 90 to 99 per cent. of protein, and is insoluble in water. It is administered in bouillon, milk, cocoa, and soup. The quantity to be given should be boiled with a small portion of the nutrient vehicle in which it is to be taken, and then mixed with the entire amount.

Salvatose, a French preparation, is a pure protein product. It is seldom used.

Fersan contains 80 to 90 per cent. of organically combined soluble protein. Fresh ox-blood mixed with twice its volume of a 1-per-cent. solution of sodium chlorid is centrifugalized, completely separating the serum containing the metabolic products; the corpuscles are then dried *in vacuo*, and powdered. *Fersan* is a dark brown, odorless powder with a slightly acid taste, soluble in water, and containing a large percentage of iron and phosphorus. The phosphorus is present in complete organic combination, and the iron almost entirely so. The preparation is an iron albuminate that calls for no digestive activity on the part of the stomach. It is not coagulated in the stomach, and is completely absorbed by the intestine. The dose is three to six teaspoonfuls a day, in milk.

Peptones.—Peptone preparations are now but seldom employed. Their nutritive value is due chiefly to the albumoses they contain. Laboratory experimentation and clinical experience have shown that, in order to obtain sufficient nourishment from the peptone preparations, unduly large quantities must be ingested. Peptones have, as a rule, a disagreeable taste. In large doses they tend to produce diarrhea. Among the most satisfactory preparations of this class the following may be briefly mentioned: The meat solution of Leube-Rosenthal contains 9 to 12 per cent. of soluble protein and 1.8 to 6.6 per cent. of peptone. Peptone chocolate contains only 6 per cent. more protein than the ordinary cocoa. Denayer's fluid meat peptone is merely a strong beef tea, pleasant to the taste, used principally as a stimulant, containing 1.5 per cent. peptone and 10.5 per cent. albumoses. Koch's peptone contains 18.8 per cent. peptone, 16 per cent. propeptone, and 1.4 per cent. insoluble protein. Cibil's peptone contains 28.1 per cent. peptone and 5.8 per cent. albumoses.

Among the artificial food preparations made in the United States we have the following, with their nutritive values, and a comparison with cow's milk, as determined by the Council of Pharmacy and Chemistry of the American Medical Association:

Name of substance	Name of manufacturer	Carbo- hydrate	Protein
1. Carpanutrine	John Wyeth & Bro.	5.34	4.28
2. Carpanutrine	John Wyeth & Bro.	5.78	6.24
3. Liquid peptones	Elb Lally & Co.	6.05	4.50
4. Liquid peptones with Creosote	Elb Lally & Co.	13.47	3.84
5. Liquid peptonoids	Arlington Chemical Co.	10.57	4.93
6. Liquid peptonoids	Arlington Chemical Co.	11.53	4.53
7. Predigested Beef	H. K. Mulford Co.	4.37	2.38
8. Predigested Beef	H. K. Mulford Co.	4.55	2.59
9. Nutrient Wine of Beef Peptone	Armour & Co.	15.43	0.64
10. Nutrient Wine of Beef Peptone	Armour & Co.	15.57	0.43
11. Nutritive Liquid Peptone	Parke, Davis & Co.	12.89	1.86
12. Nutritive Liquid Peptone	Parke, Davis & Co.	13.19	1.16
13. Panopeptone	Fairchild Bros. & Foster	11.92	6.38
14. Panopeptone	Fairchild Bros. & Foster	10.05	6.33
15. Peptonic Elixir	Wm. Merrell Chem. Co.	11.46	2.54
16. Tonic Beef S. & D.	Sharp & Dohme	2.36	3.40
17. Tonic Beef S. & D.	Sharp & Dohme	2.22	3.28
18. Liquid Peptone	Stevenson & Jester Co.	0.55	1.81
19. Cow's Milk (3.8 per cent. fat)	-	4.80	3.50

There are no fatty substances in these products; their food value from this point of view is therefore a negative quantity. They all contain alcohol; the proportion ranges from 14 to 23 per cent. The printed matter distributed by some manufacturers leads the physician to believe that these preparations contain sufficient nutritive material to maintain the normal nutrition of the body. The average quantity that can be taken daily ranges from 60 to 150 Cc. (2 to 5 ounces), the total available calories of which, based on the protein and carbohydrate bodies, varies from 9.8 to 110.5. Adding to these figures the amount of energy represented by the alcohol, in each case, the total available calories will vary from 55 to 229.5. The number of calories required per diem by a man doing very moderate work approximates 3000. In sickness the amount required is not so great, but on the average should not fall much below 1500 calories for the twenty-four hours. This consideration alone shows the fallacy of the representation that any of the artificially prepared foods above mentioned will enable the patient to dispense with other nourishment.

The report of the Council of Pharmacy and Chemistry goes on to say:

"In order to get a fair conception of the actual food value of these various preparations, it is desirable to make some comparison which can be readily comprehended by every physician. The amount of good milk necessary each twenty-four hours to sustain the vitality of a patient during a serious illness is not less than 64 ounces, or approximately 2000 Cc. (2 quarts). The food value in calories represented by this amount of good milk may be placed at 14.30. This includes not only the protein and carbohydrate matter, but the fat as well. By comparing this available potential energy with the total energy available in the predigested foods under consideration, it can be readily seen that if a physician depends on the representations made by some of the manufacturers, and feeds

his patient accordingly, he is resorting to a starvation diet. The largest number of available calories, including alcohol, present in any of the recommended daily doses is less than one-fifth of the number of calories represented by 2000 Cc. of milk; and the calories represented by the daily dose of the preparation poorest in food products is only one-twenty-fifth of the amount present in 2000 Cc. of milk. These figures tell their own story.

"Making 2000 Cc. of milk the basis of calculation, and estimating the amount of the various preparations required to yield this number of calories, it is found that the quantity to be administered daily to supply 1430 calories, including alcohol, varies from 716.2 to 1506.2 cubic centimeters (or approximately one to three pints). In many cases the amount of alcohol exhibited by these quantities would keep the patient in an alcoholic stupor continually. The cost necessary to supply this energy varies from \$1.48 to \$3.39. Compare these prices with the cost of two quarts of milk. Is further comment necessary?

"The average number of calories represented by 500 grams of these products as proteins and carbohydrates is 260.6. The total average caloric value of the same amount of these foods is 802.4.

"The number of calories represented by good brandies or whiskeys, containing 45 per cent. of alcohol, is 1575. In other words, the average caloric value of these preparations is approximately one-half that contained in either good brandy or whisky. From this it must not be concluded, however, that equal quantities of brandy or whisky are twice as valuable as the medicinal foods, because the medicinal foods contain some material which can be utilized in building tissue, which is not the case with either whisky or brandy.

"From the above it can readily be seen that not only is the patient receiving a starvation diet when the physician resorts to these preparations, but the unfortunate sick are also compelled to pay exorbitant prices for the amount of actual nutritive matter received.

"It is urged in justification of the use of preparations of this class that they contain constituents not found in our ordinary foods and in a more perfectly assimilable condition. As pointed out above, these so-called predigested foods contain no fats; the carbohydrates in them are the ordinary sugars present in our common foods, while the proteins belong to the peptone or albumose class. It is for these latter that the greatest claims are made, but even here no value can be pointed out not found in whey, peptonized full milk, or peptonized skimmed milk.

"There is likewise another point of considerable importance to consider in this connection. The terms peptone and albumose include bodies of very uncertain composition, and their suitable-

ness as food substances depends largely on how they are prepared. Animal experiments have shown that nitrogen equilibrium may be maintained, for a time at least, by the use of enzymic hydrolytic products of the proteins, even where the hydrolysis has been carried far beyond the so-called peptone stage, but it appears to be likewise true that the mixtures secured by acid or high temperature steam hydrolysis have no such value. Some of these, indeed, may exhibit a toxic behavior. This is true in particular of some of the commercial varieties of peptone, and until more is known of the source of the bodies of protein character employed in the make-up of these 'predigested' mixtures it is unwise to assume anything concerning the food value of the nitrogen compounds found in them by analysis or even to dignify them by the name of foods."

Elsall, commenting upon this report, argues against the use of proprietary foods. The development of a moderate degree of skill and resource in the use of simple and comparatively cheap home preparations will obviate any tendency to use the proprietary article. This writer cites instances where patients virtually starved to death through the mistaken belief of the physician that they were receiving sufficient nutrition from the much-vaunted proprietary food. A very important disadvantage of these foods is their alcohol content. This evil is dwelt upon in the Council's report.

Preparations from Vegetable Protein.—Among these we have:

Roborat, obtained from grain seeds, wheat, corn, and rice. It is a fine, yellowish-white, odorless, tasteless powder, only slightly soluble in cold water. It contains 83 per cent. of vegetable protein. This preparation is fairly well assimilated. It has been found of value in the treatment of gastric ulcer, atony, dilatation, erosions, enteritis, and chlorosis associated with gastro-intestinal disease. It may also be administered as a nutritive enema. *Roborat* may be given in milk or water.

Aleuronat flour is prepared from gluten. It contains 82 to 86 per cent. of vegetable protein; is almost tasteless, and is insoluble in water.

Mutae is a vegetable casein prepared from leguminous seeds. It is not expensive.

Food Preparations from Milk Protein.—These preparations contain the casein of milk and are for the most part useful foods.

Nutrose is casein sodium, a white, odorless, tasteless powder containing 85 to 90 per cent. of protein. It is soluble in warm water. *Nutrose* is almost completely absorbed by the small intestine. The casein constituent does not give rise to decomposition in the intestine. *Nutrose* has been employed in all kinds of gastro-intestinal diseases in which a mild, non-irritating and nutritive food is indicated.

Eucasin, casein ammonium, is an odorless, tasteless powder containing 85 to 90 per cent. of protein; it is soluble in water.

Sanatogen contains 95 per cent. casein and 5 per cent. glycerophosphate of sodium; the insoluble casein has been transformed by the glycerophosphate into a compound soluble in water. *Sanatogen* is well borne by patients suffering from gastric ulcer, gastritis, or acute intestinal catarrh. The dose for adults is one to three tablespoonfuls three times a day. Cow's milk contains all the ingredients claimed for *sanatogen*. Ten cents' worth of cow's milk will give the same number of calories as three dollars' worth of *sanatogen*, and the latter has no more favorable effect than cow's milk.

Plasmon consists of protein obtained by a mechanical process from skimmed milk. It is a milk-white, tasteless powder containing 74.5 per cent. of protein. *Plasmon* is easily soluble in hot water, and is almost completely absorbed in the intestine. Concentrated solutions curdle on cooling. *Plasmon* is useful in the treatment of gastric ulcer and intestinal catarrhs. It may be taken in connection with a variety of foods, as *plasmon-chocolate*, *plasmon-cocoa*, *plasmon-beef*. It is a good food, as well as the cheapest of the casein preparations.

Milk somatose is prepared from the casein of milk, and contains 5 per cent. of tannin in chemical combination. It is a yellowish-brown, odorless, almost tasteless powder, soluble in hot water. *Milk somatose* is non-irritating, and may be employed with advantage in the treatment of chronic intestinal catarrh; it is likewise useful in the treatment of dysentery and intestinal tuberculosis. The dose is four teaspoonfuls daily. (See page 277.)

Glabon is a derivative of casein obtained by breaking up nucleoprotein by means of alkalis.

Galactogen is prepared from milk; is completely soluble and easily digested. It contains 70 per cent. of protein, and is agreeable to the taste and pleasant to take in the form of *galactogen-chocolate* (20 to 22 per cent. soluble protein) or *galactogen-cocoa* (30 to 32 per cent. soluble protein).

Mammala is cow's milk modified by removing a part of the cream, adding milk-sugar, and drying *in vacuo*.

Nutritive Substances from Egg Protein. *Nutritive-Heyden* is prepared from the whites of fresh eggs, and contains 90 per cent. of protein. It is soluble only in hot water. The dose is three to four dessertspoonfuls, in cocoa, soup, or milk. It may be given also as an enema.

Prologen is a formaldehyde protein prepared by the action of formalin on egg protein. It is not much used.

Preparations from Carbohydrates. These preparations are better adapted than protein compounds to increase the nutritive value of certain foods, especially soups, and to serve as substitutes for ordinary diet.

Finely Divided Flours.—Hartenstein's legumins are furnished in four mixtures:

I . . .	27 per cent. protein;	62 per cent. carbohydrates.
II . . .	21 " "	68 " "
III . . .	18 " "	69 " "
IV . . .	15 " "	72 " "

Knorr's Flours (oat, barley, rice, bean, lentil, pea) contain 7 to 25.5 per cent. protein, 57 to 79 per cent. carbohydrates.

The meals belonging to this class are rolled oats and oatmeal—12.67 per cent. protein, 63.8 per cent. carbohydrates.

The utility of these preparations is great, compared with that of corresponding products in common use.

Dextrinated Flours.—In these flours the carbohydrates are dextrinated. To this group belong the extensive series of infant's flours—Carnrick's lactated milk, malted milk, Ridge's, Wagner's, Mellin's, Nestlé's, Eskay's, Allenburys', and Imperial Granum. Owing to the fact that it contains dextrinated starch, malt extract also belongs to this group. Malt extract is a well-known product of germinating barley; it contains, condensed to a syrupy consistency, 50 to 55 per cent. of sugar, of which 10 to 15 per cent. is dextrinated soluble starch. Loefflund's malt soup is a good food. It contains 57 per cent. of maltose and 12.4 per cent. of dextrin. Maltose buttermilk, containing dextri-maltose and wheat flour, is a serviceable article of diet in cases of disturbed nutritional balance with excess of fat. The malt extracts of Keppler, Trommer, maltine, and malt beers have no particular value as food agents.

Mixed Nutritive Preparations.—The preparations above mentioned contain not only carbohydrates, but also more or less protein. Recently mixtures of carbohydrates and proteins, the latter partially treated with ferments, have been offered. Among such preparations we have:

Hygiama, consisting of condensed milk, especially prepared cereals, and fat-free cocoa. It contains 22.8 per cent. protein, 61.6 to 63.32 per cent. carbohydrates. Two dessertspoonfuls with one-quarter liter (8 ounces) of milk, three or four times a day, constitute the dose. Hygiama tablets may be eaten without any further preparation.

Odda is a mixture of yolk of egg, cocoa fat, whey, dextrinated flour and other carbohydrates. It contains 16.56 per cent. protein and 18.14 per cent. carbohydrates.

Protein-milk-salt-cocoa, a new compound originated by Simon, of Carlsbad, belongs to this division. It is a cocoa containing only 15 per cent. of fat, combined with 37.23 per cent. of protein predigested with ferments, and 7.61 per cent. of nutritive milk salts. Up to 74 per cent. of the protein of this cocoa is digestible, and the cocoa itself contains more digestible protein than an equal weight of raw beef. The taste is very pleasant. This cocoa is

particularly useful in cases of chronic gastro-intestinal disease, especially as its cost is comparatively low.

Rucahout is a compound of chocolate, sugar, and Arabian meal, very nutritious and agreeable. It is useful in all gastro-intestinal diseases. Two to four teaspoonfuls are added to milk, which is then allowed to boil for seven or eight minutes.

Acorn-cocoa consists of pure cocoa deprived of a large part of its fatty matter and combined with a soluble extract of roasted acorns free from cellulose. A small amount of roasted flour and sugar is added. On account of its astringent properties it is valuable in gastric catarrh and all forms of enteritis.

Preparations Containing Fat.—*Russell's Emulsion* contains beef suet, cocoanut oil, peanut oil, and cottonseed oil, to the extent of 42 per cent. of its volume.

Nutrole contains 40 per cent. of mixed animal and vegetable oils, emulsified with fresh eggs.

Sevetol is a natural emulsion of mixed fats with proteins and carbohydrates. The fats are butter, beef fat, olive oil, lard, and peanut oil; these make up 30 per cent. of the whole mixture.

Cod-liver oil contains a considerable proportion of fatty acids with biliary elements. It is rich in vitamin, the crude oil, more so than the refined. It is converted by means of the bile into a very fine emulsion, and is most thoroughly absorbed. Its taste is exceedingly repugnant. Cod-liver oil in elastic gelatin capsules can sometimes be taken by those who cannot take the oil unmasked.

Oil of sesame is more agreeable to the taste than cod-liver oil, and cheaper.

Liparin is a cod-liver oil substitute, consisting of a mixture of 94 parts fine olive oil and 6 parts oleic acid. It has a pleasant taste and causes no subjective discomforts.

Mering's "Kraft" Chocolate contains 72.44 per cent. fat to which oleic acid has been added. It is very easily digested.

Milk Preparations.—*Vegetable Milk* is made of nuts and milk of almonds (10 per cent. protein, 25 per cent. fat, 38.5 per cent. sugar).

Pfund's Cream Protein Mixture is a mixture of various kinds of proteins with milk-sugar, cream, and water.

Gärtner's Fat Milk and *Voltmer's Mother's Milk* are fat milks digested with pancreatic juice; they are very similar to human milk.

Kefir and *koumiss* are preparations of milk which have been subjected to fermentation (see page 164).

Stimulating Preparations.—*Liebig's Meat Extract* contains the extractives of meat, the meat bases xanthin and creatin, and inorganic salts.

Toril Meat Extract, *Beef Tea*, and *Valentine's Meat Juice* are poorer in extractives than Liebig's meat extract.

Brand's Essence of Beef, Meat Juice, Fluid Meat, and Borril, much used in England, contain smaller quantities of extractive than Liebig's extract of meat.

Karno is less nutritious than Liebig's extract of meat.

Maggi's Condiment is cheap and good. *Maggi's Bouillon* is also to be recommended as a stimulating preparation.

Composition and Relative Values of Meat Extracts.—The Bureau of Chemistry of the Department of Agriculture, in its Bulletin No. 114, has given valuable data regarding the commercial meat products. The preparations taken up are divided into three general classes:

1. Solid and fluid meat extracts.
2. Meat juices.
3. Miscellaneous preparations.

Meat extracts are not to be considered as foods, and should therefore not be advertised as such—a conclusion which the government officials have arrived at, and which they have stated as follows:

"It seems to be the consensus of opinion among scientific investigators who have studied this question that the food value of these meat extracts is rather limited, and although they are a source of energy to the body they must not be looked on as representing in any notable degree the food value of the beef or other meat from which they are derived. When prepared under the best possible conditions a commercial meat extract is of necessity, in order that it may not spoil, deprived of the greater part of coagulable proteins, which constitute the chief nutritious elements of the juice."

The physician should realize that in prescribing preparations that have but little food value he may actually starve the patient. According to the high authority quoted, the claims of the manufacturers in regard to the food value of "meat extracts" and "meat juices" are ridiculous. The therapeutic uses of these preparations are therefore limited. It has been claimed that such substances stimulate appetite and the nervous system. They may stimulate the appetite, but their effects upon the nervous system are open to question.

The belief of many people that bouillon cubes are concentrated meat extracts and of high nutritive value, has been shattered by a recently issued bulletin of the Department of Agriculture, which says that, while they are valuable stimulants or flavoring agents, they have little or no real food value and are expensive. The ordinary commercial bouillon cubes consist of from 49 to 72 per cent. table salt. As they range in price from 10 to 20 cents an ounce, purchasers of these cubes are buying salt at a high price. The cubes do contain a small amount of protein in addition to their

stimulating ingredients, and the makers of most of the cubes make no advertised claim that they are concentrated beef broth or essence. However, many housewives believe that they are and that they possess high nutritive value, especially for invalids. This is not the case. The fact that each cube makes a cup of broth misleads the housewife into believing that she is securing meat extract cheaply when really she is buying it in an expensive form.

CHAPTER IX.

LAVAGE OF THE STOMACH.

LAVAGE, or the washing out of the stomach, is not practiced nearly so often as it was at one time. Our knowledge of the exact course of many diseases of the stomach, and of the pathologic changes accompanying them, has advanced. Lavage was formerly used in the treatment of many conditions in which, with our more accurate knowledge, it has now been discarded.

Indications.—Lavage is always indicated in stenosis of the pylorus with dilatation—in fact, in any obstruction of the digestive tract which produces a stasis of the stomach contents with fermentation and putrefaction. Good results are not attained in the treatment of simple atony by means of lavage, because in this condition we are dealing with a retarded peristalsis and not with a direct obstacle to the passage of food into the duodenum; the washing-out process does not tend to remove the cause, but involves the danger of overdistention of the relaxed gastric walls, which is apt to be harmful.

In certain conditions lavage is of inestimable value; it is indicated:

1. In those cases of poisoning in which the tube can do no damage. There is always danger of perforation when the poison has been an escharotic or caustic. In morphin poisoning the tube should be used even if the drug has been taken hypodermically, since much of the morphin injected hypodermically is found in the stomach within an hour after the injection.
2. In cases of uncontrollable vomiting, as in intussusception or intestinal obstruction. There have been cases reported in which lavage so relieved abdominal distention near the obstruction as to result in almost immediate recovery. Stercoraceous vomiting always demands lavage, no matter what the cause may be.
3. In cases of gastritis with the production or presence of large quantities of mucus.
4. In dilatation with stenosis of the pylorus. Here fermentation and putrefaction can be inhibited by lavage. These are the cases concerning which Kussmaul originally called our attention to the value of stomach washing.
5. In acute postoperative dilatation.
6. Before any operation on the stomach or intestine is performed.
7. In vomiting following any operation on the stomach or intestine.
8. To obviate postoperative vomiting after an anesthetic.
9. In intestinal paresis following operation.

10. Lavage with ice-water in hemorrhage caused by gastric ulcer (Ewald). Lavage, carefully applied, in severe hemorrhage from gastric ulcer, is the most expedient means of treatment (Kaufmann).

11. In meteorism of typhoid fever it is frequently of great benefit.

12. In gastric tetany.

13. In vomiting in cases of peritonitis.

14. In acute gastritis due to improper eating, and in convulsions following overfeeding.

15. In cicatricial closure of the pylorus, as a palliative measure until operation is performed.

16. In hematemesis following stomach operation, cautiously. The stomach may be distended with fluid and blood, removal of which will allow it to contract and thus stop the oozing of blood (Mayo).

17. In diabetes mellitus (Sawyer).

18. In selected cases of nephritis when urea is being eliminated through the gastric mucous membrane.

19. In eclampsia.

Contra-indications.—Lavage as well as the use of the tube for diagnostic purposes is contra-indicated:

1. In those cases of gastric disease, for the most part of sudden onset, which have not attained any degree of chronicity and where the diagnosis is apparent from the symptoms and history of the case.

2. Where the retching and vomiting are apt to offset any good that may be derived from the use of the tube either for diagnostic purposes or for lavage.

3. In marked prostration, no matter what the cause.

4. In broken compensation in heart disease, angina pectoris, or advanced degeneration of the heart muscle, and in cardiac neuroses, aneurysm of the aorta, and marked cases of arteriosclerosis.

5. In hemorrhages of recent occurrence, as in apoplexy, pulmonary, renal, gastric and rectal hemorrhages.

6. In pulmonary tuberculosis, emphysema, and severe bronchitis.

7. In neurasthenia, hysteria, and epilepsy.

8. In advanced cachexia.

9. In continued and remittent fever.

10. In pregnancy.

11. In gastric ulcer when hematemesis has been recent or when blood has been found in the stool; in carcinoma of the pylorus accompanied by the classic symptoms of carcinoma; in gastric or intestinal diseases accompanied by acute fever; and in cases in which the gastric mucous membrane is easily irritated so that bleeding results upon the passage of the stomach tube.

Any rules which may be laid down in regard to the use of the stomach tube are at best but general. The good judgment of the physician must always be superadded, whether the question be one

of diagnosis or treatment, inasmuch as there may be conditions present which outweigh all stated rules on the subject.

Technic.—Lavage consists in the washing-out of the stomach by means of a simply constructed apparatus—a stomach tube (Fig. 16, A) connected with a glass funnel or irrigator, with a piece of glass tubing between. The stomach tube (Fig. 17) should have two lateral oval openings near the point; and the point should be solid and closed to prevent the collection of material below the open-

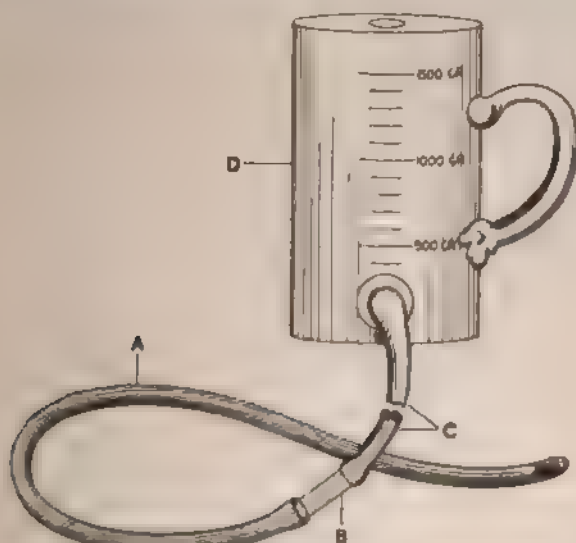


FIG. 16.—Apparatus for gastric or colonic lavage. A, stomach tube; B, glass tube; C, rubber tube connection, D, glass irrigator.

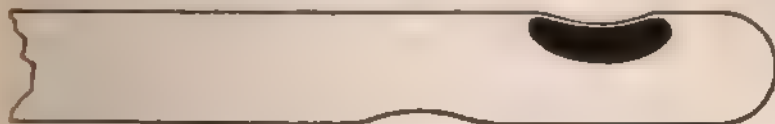


FIG. 17.—Stomach tube showing elongated lateral openings.

ings. The edges of the openings should be smooth and rounded, since otherwise particles of mucous membrane may be caught and torn off. The tubes for adults should be large, averaging Nos. 36 to 38.¹ The funnel of the lavage apparatus (Fig. 18) should have a capacity of one-half to one liter. The small end of the funnel must be large enough to permit the passage of food. The rubber connecting tube must be of the same caliber as the stomach tube, and long enough

¹ Some confusion has resulted from the fact that there are three standards of measurement—American, English and French. To obviate error the American Medical Trade Association has adopted the French standard, so figures designating the sizes of tubes will be in the French or standard metric scale.

to reach from the patient's mouth to the floor of the room. A large glass irrigator is probably better than the glass funnel; its capacity should be at least 1500 Cc. (3 pints). The irrigator is provided with a handle, and has a hole near the brim by which it may be suspended on a hook (Fig. 16). The lower or outflow opening should correspond in diameter to the caliber of the stomach tube.

Lavage with this simple apparatus is accomplished as follows: The patient should be impressed by his physician with the necessity of the washing-out process. He should be seated in a comfortable position, with the body inclined slightly forward, and instructed to breathe regularly and deeply. He is taught to make energetic movements of swallowing at the command "swallow." Artificial teeth should be removed before lavage is begun. The patient's

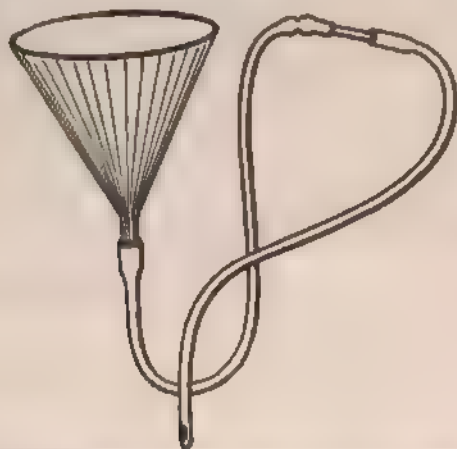


FIG. 18.—Stomach tube showing funnel connections.

hands may be employed in holding a pus basin or other receptacle for the purpose of cleanliness, and in this way any interference on his part may be obviated. The stomach tube should be moistened with water, not oil, and directed over the dorsum of the tongue. When the end of the tube reaches the posterior pharyngeal wall, deglutition begins. The tube slides easily over the cricoid cartilage into the first section of the esophagus. When this point is reached, it is easy to pass the tube on into the stomach. (The slight irritation effected by moving the tube up and down is sufficient to cause the evacuation of large quantities of stomach contents, especially if aided by pressure on the abdominal muscles on the part of the patient.) A pint of lukewarm water should be placed by an assistant in the irrigator or funnel, held low. The tube of the irrigator is meanwhile stopped by means of the fingers or clamp at a short distance from the free end, and connection is duly made with

the glass joint and the stomach tube in position. The irrigator is then raised until nearly the whole quantity of water has passed into the patient's stomach. A small quantity of water should be left in the irrigator to prevent the entrance of air into the tube. The irrigator is now lowered to the floor of the room, so that the stomach contents, including the water, may be siphoned off. It should be held in such a manner that the outflowing liquid may be visible. After noting the difference between the outflowing fluid and the clear water that entered the stomach, the contents of the irrigator may be emptied in a convenient receptacle. More water is allowed to enter the stomach, and the process of lavage continues by alternately raising and lowering the irrigator until the water comes from the stomach clear. When lavage has been completed, the stomach tube should be detached from the irrigator and rapidly and gently withdrawn from the patient's stomach. It is important to disconnect the irrigator and tube; otherwise, with the former resting on the floor, suction produced by the siphon effect would tend to invaginate the mucous lining of the stomach into the lateral openings of the tube and thereby injure the stomach wall.

In the absence of an assistant, the physician should fill the irrigator with the required quantity of water before commencing the operation. In order to keep the tube of the irrigator free from air, it should be compressed by means of a large tube-compressor near the glass connection as soon as the water begins to flow through. The introduction of the stomach tube follows. The patient is directed to keep the tube steady with one hand at his mouth, while with the other he holds the basin. With the irrigator resting on the floor, the physician may connect it with the stomach tube, loosen the tube-clamp or compressor, and elevate the irrigator.

I have found my improved stomach tube and bulb (Figs. 1 and 2) for removal of the stomach contents a simple and practical apparatus for gastric lavage. A bulbful of water can be easily utilized as described on page 71.

Patients to whom stomach lavage must be administered regularly and over a long period of time can be taught to carry out the operation without the aid of a physician. Autolavage is a form of stomach irrigation which has been called physiologic in order to distinguish it from the kind I have just described; for this the use of the stomach tube is not necessary. It is sufficient that the patient drink four to eight ounces of the irrigating fluid and then lie down on his abdomen, supported on a somewhat hard, resisting surface, across the bed or on the floor. In this position let him breathe as deeply as possible. Fifteen to twenty deep inspirations are sufficient to drive the contents of the stomach through the pylorus. This procedure may be repeated as often as necessary.

As a rule the patient may rest on his abdomen for five minutes, taking from time to time a number of deep inspirations. It has been proved that in this way the stomach may be cleansed quite as effectively as by the introduction of the stomach tube, provided the pylorus be not occluded. This method has a considerable advantage over the other, for by it the nourishment, as prepared by the stomach, is not lost, but follows the physiologic path. Besides, the patient will submit much more readily to it than to the manipulations of lavage with the stomach tube. In order to obtain the maximum effect from this method of autolavage, we must strive by all means at our command to free the pylorus from all obstacles that interfere with its proper function. This is partially achieved by administering the fluid lukewarm.

Some patients may be taught to use the stomach tube themselves with the aid of some member of the household. None but the best apparatus should be employed. After use it should be thoroughly cleansed by means of hot water. In lavage, whether the patient uses the apparatus without the aid of the physician, or whether the physician performs the operation upon a passive patient, the simple apparatus described will be found adequate for all purposes.

Fig. 19 illustrates the apparatus designed by Friedlieb on the principle of suction. This instrument was designed to facilitate the removal of obstructing particles from the stomach tube by aspiration by means of a rubber bulb. The apparatus of Strauss (Fig. 20) accomplishes the same purpose by means of a double bulb. Both these instruments, in the opinion of the author, are unnecessary, inasmuch as clogging of the tube may be prevented by raising the irrigator of the apparatus described above, and thus forcing the tube clear by water pressure.

In cases where the stomach is greatly dilated it is frequently impossible to wash it out at one sitting. In such cases lavage may be better accomplished with the patient in a recumbent posture. With the patient seated, the thoroughness of lavage may be promoted by pressing or kneading the hypogastric region after the water has been introduced into the stomach.

In cases where, owing to irritability of the fauces, it seems impossible to introduce the stomach tube, the difficulty may be overcome by painting the fauces with a 5-per-cent. solution of cocaine or beta-eucain. Another effective and an entirely safe method of preventing nausea from the introduction of the stomach tube is to freeze two or three inches of the extremity of the tube just before introducing it, the object being to secure light temporary anesthesia of the fauces and pharynx by means of the cold rubber. In this way cold is applied exactly where anesthesia is needed, and the irritability is overcome. Thus the tube may be introduced for the first time with practically no gagging, straining, or nausea.

The extremity of the tube may be frozen by a few moments' spraying with ethyl chlorid. The tube, of course, may be chilled in other ways, but the ethyl chlorid is convenient and efficient. The tube has been found not to stiffen markedly under the influence of the extreme cold, so that no trauma from the frozen rubber occurs. By the time the tube reaches the cardia its low temperature is sufficiently modified to obviate danger to the gastric mucosa, even though it be allowed to remain in the stomach for some time.

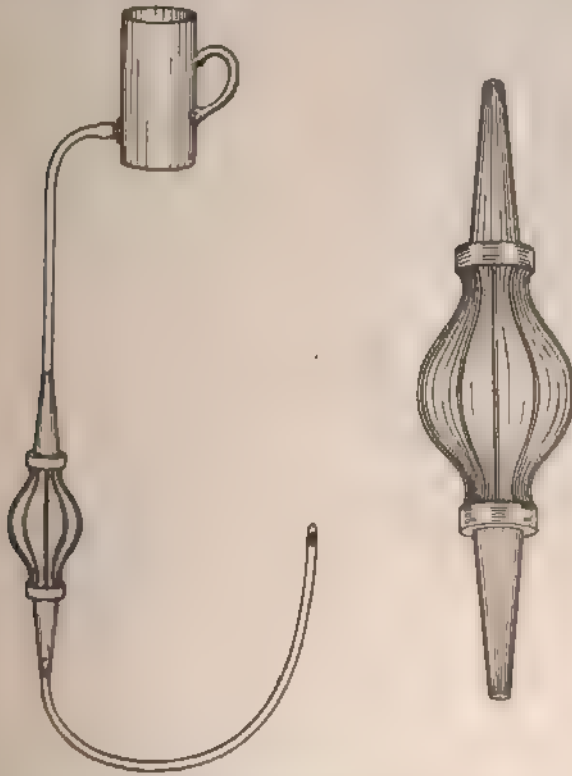


FIG. 19.—Stomach tube with suction bulb. (Friedlieb.)

The process of washing out the stomach is not attended with any danger. Temporary cessation of respiration, of reflex origin, occurs in many patients at the first introduction of the tube. This, however, should not occasion anxiety on the part of the physician, since it usually passes off readily. Should the patient become alarmed and attempt to pull out the tube, an emphatic request to "breathe deeply" will overcome his fears and make possible the complete introduction of the tube. Where paroxysms of cough, severe and protracted, supervene, the operation of lavage should be interrupted before completion.

In the presence of gastric hemorrhage, such as occasionally occurs in cases of carcinoma or ulcer, lavage is contra-indicated. It is rarely employed in cases of carcinoma except as a palliative measure when the pylorus is obstructed. In gastric ulcer it is apt to do a great deal of harm, and should never be employed when there is any indication of gastric hemorrhage. In cases of nervous dyspepsia lavage sometimes transforms the patient into a gastric hypochondriac, a most lamentable condition. In the majority of nervous cases lavage is contra-indicated. Surface hemorrhage may take place in catarrh due to gastritis; when such hemorrhages are of a pronounced character the irrigations should be discontinued.

Not over 2 per cent. of cases of gastric disease, or of patients presenting symptoms suggestive of gastric disease, require lavage as an element of treatment.

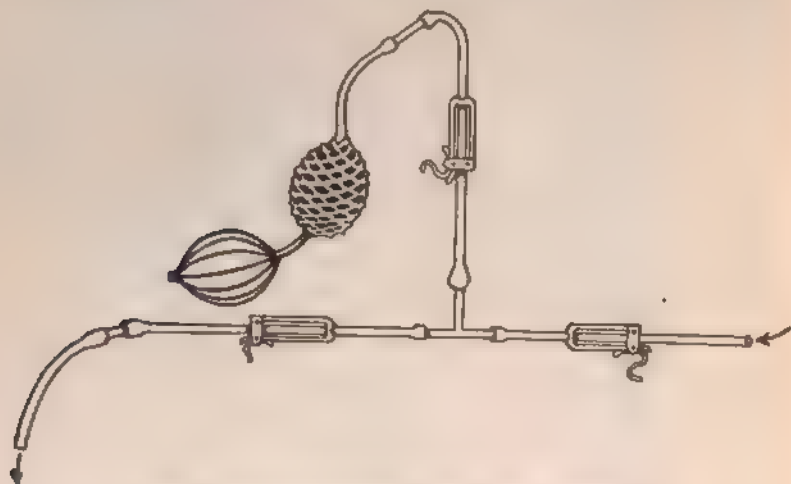


FIG. 20. Suction tube with double bulb. (STRUVE.)

Gastroenterologists differ in their views regarding the time stomach irrigations should be administered. I consider it advisable in cases where there is no engorgement, for example in cases of chronic gastritis, when we wish to remove mucous secretion, to perform irrigation in the morning while the stomach is empty. In cases of stenosis of the pylorus, with stagnant masses of food in the stomach, the best time for lavage is in the evening shortly before the evening meal.

The duration of the treatment must be determined in each case by the conditions present. In cases where it is impossible to determine this point, as in inoperable carcinoma, it is advisable to have the patient wash out his own stomach. A medicated lavage may follow the cleansing lavage. The indications for the different kinds of lavage are given under the respective diseases.

In lavage preliminary to surgical operation on the stomach, care should be exercised that no water remains in the viscus. In lavage kept up, as described, until the washings return clear, a further quantity of water can be dislodged by placing the patient in the Trendelenburg position; the flow will continue until the tube is withdrawn from the cardiac orifice and the stomach is entirely emptied.

THE STOMACH DOUCHE.

Douching of the stomach should be employed only when the viscus is empty. The sole object is to irrigate the mucous membrane, either with plain water or with medicated solutions. The douching may be performed by means of Rosenheim's tube (Fig. 21). This instrument consists of a stomach tube having at its gastric extremity a number of small openings, from one to two millimeters in diameter. Water is permitted to flow through the tube into the stomach so that all parts of the gastric mucosa are irrigated through the numerous small openings. In the process, however, one or more of the fenestræ are frequently blocked by mucus.



FIG. 21.—Perforated tube. (Rosenheim.)

In Richter's method of removing mucus by irrigation, an ordinary stomach tube is introduced to the extent of 40 centimeters, or to the cardia of the empty stomach. The irrigating fluid, under pressure, is allowed to pour into the stomach, douching the collapsed walls. While a small quantity of water yet remains in the irrigator the tube is pushed into the stomach so that the fenestræ become immersed in the water there; the irrigator is then lowered to the floor and the contents are siphoned out. The tube is then withdrawn to the cardia, and the process is repeated as often as necessary to cleanse the stomach of the mucous secretion.

Einhorn's apparatus (Fig. 22) consists of a tube (*A*) about 60 cm. in length and 1 cm. in diameter, having at the gastric extremity a cylinder of hard rubber shaped like a capsule (*B*). This capsule has numerous minute openings, and at the lower end a larger round aperture. Within the hard rubber capsule is an aluminum ball (*C*), which acts as a valve and closes the opening in the extremity of the capsule when the tube is introduced and the irrigating fluid forced into it. The water enters the stomach by way of the small openings. The return flow, however, forces the ball from the lower opening, and the entering liquid keeps this opening clear until the stomach is completely emptied. The

defects of the Rosenheim tube are remedied in Einhorn's apparatus. Preparatory to the entrance of the irrigating fluid, Einhorn's tube should be introduced only a short distance below the cardia; but to facilitate the return flow of water and mucus it should be pushed in 10 to 12 centimeters farther.

Turek has devised a double-flow stomach douche, consisting of two tubes cemented together; one tube is longer than the other, which enables it to reach the fundus while the shorter tube is

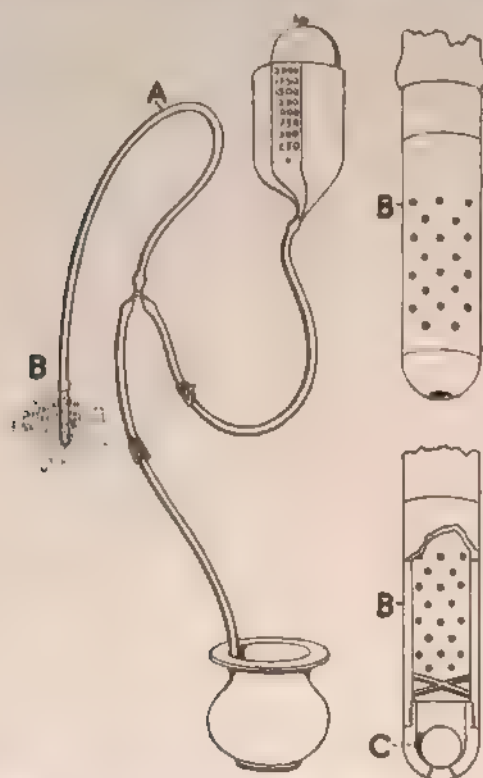


FIG. 22.—Apparatus for stomach douche. (Einhorn.) *A*, stomach tube, *B*, hard rubber capsule; *C*, aluminum ball.

near the cardia. The latter has at its end a metal ball, finely perforated; the water passing through acts as a fine needle spray or douche on the mucous membrane of the stomach. The longer tube carries the water back, so that the stomach is not distended with too great a quantity of water at any one time.

Chase has devised an improved tube (Fig. 23), by means of which (1) the gastric contents can be removed by aspiration, (2) the stomach washed or douched, (3) and inflation of the stomach effected, without making a connection or disconnection of the

apparatus and without the use of stopcock or shut-off. By substituting a "Rosenheim" douching tube the stomach may be douched as recommended by Rosenheim. Chase's apparatus consists of (1) an Ewald stomach tube proper, 30 inches long,

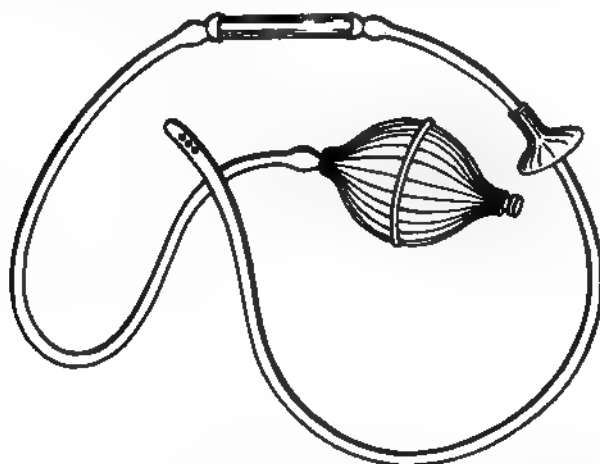


FIG. 23.—Stomach tube. (Chase.)

marked at 22 inches from its distal end with a white band; (2) an adjustable saliva shield, to prevent saliva from flowing down the tube; (3) a glass connector; and (4) a 30-inch connecting tube, to which is attached a strong valveless bulb of 3 ounces (90 Cc.) capacity.

CHAPTER X.

MASSAGE—ELECTRICITY.

MASSAGE OF THE STOMACH.

MASSAGE consists of a systematic manipulation for definite therapeutic ends. The success of the process depends upon the precise performance of certain well understood movements of the hands of the physician. The operator in applying the treatment should keep in mind the particular end to be accomplished in the individual patient. The several movements consist of various applications of rubbing, kneading, stretching, and pinching of the muscles. The two hands must be directed with intelligence and skill.

Indications.—Massage is of greatest value in diseases due to altered metabolism, and in those in which the powers of digestion, absorption or assimilation are defective. Nutrition may be profoundly influenced by regular and continued massage. Among the special indications for this mechanical treatment are:

1. *Relaxed musculature* which may be strengthened by passive exercise, and *connective-tissue adhesions* that require to be relaxed or broken up.

2. *Retention of gastric contents for an abnormally long time in the alimentary tract.* This applies more particularly to the intestine than to the stomach, where under some conditions mechanical treatment may cause direct injury.

3. *Certain forms of dilatation due to pyloric stenosis.* In the presence of marked fermentative processes massage should not be employed, owing to the possibility of propelling fermenting masses into the intestine, where the conditions for the growth and multiplication of bacteria are much more favorable than in the stomach.

4. *In certain sensory forms of nervous dyspepsia,* where sensations of pressure or pain are present, massage may be tentatively employed.

5. The mechanical treatment of the abdomen has given favorable results in cases of *primary intestinal atony and constipation* tending to secondary disturbances of the gastric function.

Contra-indications.—Massage is contra-indicated in all recent cases of ulcer with adhesions, in which cases even its cautious application may cause a perforation of the ulcer into a neighboring organ, with the well-known disastrous effects. It should not be employed in any residual inflammatory conditions of the gastro-

intestinal tract, nor in the acute inflammatory stage in which there are symptoms of meteorism or fever. It should be avoided in the presence of abdominal pain. Patients with hyperchlorhydria or hypersecretion are not to be subjected to massage, owing to the danger of inducing ulcer of the stomach. Massage is contra-indicated in dilatation of the stomach in which organic stenosis is present. If the gastric muscles are spontaneously very active, if the peristaltic movements are pronounced and frequent or sometimes as if in a tetanic condition, or if the stomach feels under the hand somewhat like a contracted uterus after birth (muscular rigidity), the massage treatment should not be employed.

Carcinoma of the stomach is always an absolute contra-indication for massage, owing to the possibility of exciting to rapid growth a tumor that has hitherto been latent. Massage in the treatment of patients above forty years of age in whom the symptoms of gastric disease have appeared suddenly, unless malignancy can be positively excluded, should be performed with great caution. Inconsiderate massage of the abdomen may stimulate a latent intestinal carcinoma to rapid growth and metastasis. Dormant gastric ulcers may be awakened by massage to harmful activity. Whenever the test for occult blood in the feces is positive, massage is contra-indicated. The physician should examine McBurney's point and the region of the gall bladder for possible inflammation before attempting massage of the abdomen. A history of gastralgia at any time, especially before or after pregnancy, increases the probability of latent gallstone disease, contra-indicating massage. There are various affections of the liver, spleen and pancreas which contra-indicate abdominal massage. In fact, abdominal pain of any kind contra-indicates it.

Massage may be applied when the stomach is either full or empty. When the stomach is filled, massage is indicated in cases of spasm of the pylorus and in mild cases of organic stenosis, the purpose being to propel the macerated food into the intestine. It should be performed three or four hours after the chief meal.

Technic.—The technic of the mechanical treatment must vary according to the object to be accomplished. When the object is passive, evacuation of the stomach contents through the pylorus, insert the right hand deeply in the loose flesh on the left side, grasping a portion of the stomach between the thumb and the four fingers, and by a pushing motion at the fold move the gastric contents toward the pylorus. The left hand advances toward the pyloric exit, beginning near the thumb of the right hand. The patient should be lying in an inclined position, the body sloping toward the right side. These movements on the part of the physician should be repeated as often as necessary. The massage movements on the full stomach should be concluded by short tapping strokes, technically known as *tapotement*. Both hands of the

operator are placed vertically, midway between supination and pronation, over the part to be treated; they are then completely pronated and the stomach is tapped with the fingers widely separated. The movements should be executed rapidly, but too great force should be avoided. *Tapotement*, as it is called, has a stimulating effect upon the musculature of the stomach.

Pétrissage is performed in the following manner: The operator stands at the right side of the patient and presses with the right hand in the gastric region in the middle line. The pressure is deep, to reach the spinal column, thereby dividing the stomach into two equal parts—one the fundus, the other the pylorus. The food mixture compressed in the pyloric half is then to be pushed toward the pylorus so that it may act somewhat like a bougie, dilating the pyloric exit.

On account of the deep situation of the stomach and the slight resistance of the deep plane on which it rests, only a limited portion of the viscus can be reached in the dorsal decubitus. For a dilated stomach the author kneads at first from left to right with patient on back, knees bent and head raised. After a few minutes he has the patient lie on the right side, and *pétrissage* is performed with both hands alternately, from pylorus toward cardia. Gentleness is necessary during the seance. The operation should last about fifteen minutes for the stomach alone, and fifteen minutes more for the intestine if there is constipation. The treatment should be given two or three hours after a meal. The beneficial effect most frequently manifests itself first by a returning appetite, then by the disappearance of the rumblings, cructations, gastric pains, headache, vertigo, etc. At the beginning the diet must be light and limited in quantity. Massage is of benefit in chronic gastritis, nervous dyspepsia, gastralgia due to neurasthenia or anemia, and pylorospasm, but it may do harm in ulcers or tumors of the pylorus. The massage movements are not always successful in expelling the contents of the stomach into the duodenum.

For improving the tone of the empty stomach, *Crédé's method* may be applied. This well-known process is employed frequently in delivering the placenta—by expression, just as a stone is removed from a cherry. By means of the expression movement in the line of the transverse axis of the stomach, we endeavor to propel the residue of gastric contents into the duodenum.

Massage movements may be facilitated by lubricating the epigastric region with pure olive oil or with glycerin. By the use of glycerin, oily stains on the clothing may be avoided. One-per-cent. salicylic acid added to the glycerin will prevent irritation of the skin.

Wegele recommends the employment of medicated lavage in conjunction with massage in various forms of chronic gastritis and in hyperacidity, for the relief of hyperesthesia of the mucous

membrane, or nervous gastralgia. The medication he employs includes physiologic salt solution; 1-per-cent. solution of ichthyol; 5-per-cent. Carlsbad salt solution; 5- or 6-per-cent. suspension of tannin subnitrate; 1- or 2-per-cent. silver nitrate solution, followed by rinsing with normal saline solution; decoctions of bitter tonics; and disinfecting solutions. The fluids are either swallowed or introduced by means of the stomach tube.

Massage of the stomach should never be delegated to a layman to perform, nor should it be undertaken by any one who is not thoroughly conversant with the principles of the treatment.

Vibratory massage is of little or no value in the treatment of diseases of the stomach. It is of value in neurasthenic conditions, when it should be applied to the spine. It should never be used directly on the stomach in any diseased condition of that viscus. The pylorus will open, according to Abrams, from pressure over the fifth dorsal vertebra. Either pressure or percussion at this location suffices.

INTESTINAL MASSAGE.

Physical exercises perform an important rôle in strengthening the abdominal walls while they add tone to the musculature of the intestinal tract.

Massage of the abdomen and intestine is recommended along with gastric massage. The purpose of abdominal massage is to strengthen the relaxed abdominal walls, stimulate peristalsis, and improve the circulation in the abdominal vessels by stimulation of the sympathetic nervous system. The technic of abdominal massage is as follows: The patient should be placed on a firm couch or table, with his head slightly elevated; the lower extremities are flexed at the hips and knees. The physician occupies a position to the right of the patient. Massage should be commenced gently, especially in the case of patients on whom it is being performed for the first time, in order to prevent rigidity of the abdominal walls which renders deep massage practically impossible. Both hands should be laid upon the abdomen and slight stroking movements made (rotating effleurage). Concentric circles should be made in the direction of the hands of the clock. The movements should be begun at the symphysis, proceeding upward and then over the entire abdomen (Fig. 24). These movements are designed to overcome the tension of the abdominal walls; in particularly stout patients the circular movements may be followed by kneading of the abdominal walls (*pétrissage*). Deep kneading of the intestine should follow, the purpose being to stimulate intestinal peristalsis and thereby loosen impacted fecal matter. Both hands should follow the direction of the intestine through the abdominal walls; zigzag movements to and fro are to be made (Fig. 25).



FIG. 24 —Abdominal massage, first movement. (Hoffa.)



FIG. 25 Abdominal massage, second movement. (Hoffa.)

Deep pétrissage should involve the whole abdomen, affecting particularly the median portion of the intestinal tract, namely, the ileum. The operator should next proceed toward the large intestine. The movements are made first by the right hand, which is dorsally flexed and placed in the right pubic fossa at the beginning of the ascending colon (Fig. 26). Pressure is made as deeply as possible, and in order to augment it the points of the fingers of the left hand should be pressed upon those of the right. The movement is first upward toward the hepatic flexure, then transversely below the arch of the ribs toward the left side, and finally downward so that the stroke penetrates deeply into the left iliac fossa. The pressure then ceases and the hands glide over the bladder back to



FIG. 26.—Abdominal massage, third movement. (Hoffa.)

the right iliac fossa, from which point the stroking of the large intestine should be repeated several times. A few rotating effleurage movements of a soothing nature should be performed. Then should follow rotating pétrissage of the large intestine. With the left hand placed over the right, the fingers of both hands should push with a rotating motion into the cecal region, the finger ends pointing toward the chest. The pressure should be light at first, but gradually increased until the whole course of the large intestine is thoroughly massaged with this rotating pétrissage.

The muscles of the intestinal tract should then be subjected to slight stimulating "tapotement." While executing these latter movements the hands should be held so that the thumb is approxi-

mated to the index finger, and the other fingers are slightly flexed. The abdomen should be slightly tapped in all directions. More vigorous tapotement may be performed with the dorsal surface of the flexed fingers, the middle finger being elevated slightly above the others. This procedure may be advantageously followed by shaking motions with the right hand placed flat on the central part of the abdomen, the fingers of the operator being spread widely apart.

In chronic constipation, by careful, continued and frequently repeated massage of the intestine the bowel may be emptied, the weakened intestinal muscles stimulated, and the secretions—nearly always deficient in this disease—brought back in normal quantity. When the patients have begun to improve, a careful and punctual habit of defecation should be inculcated, that a permanent cure may result. Before attempting to use massage for chronic constipation it is necessary to empty the bowel thoroughly by enemata, lest there be some retention of impacted feces in the colon, a condition which cannot always be excluded by a daily evacuation by means of purgatives. Should abdominal massage be applied while these impacted masses are in the bowel, inflammatory disturbances might result. A preliminary Roentgen-ray examination will reveal the exact position of the colon, enabling the operator to follow its course with certainty.

The sympathetic nerve plexuses may be reached by massage. In order to get at the celiac plexus the ends of the fingers are placed lightly upon the abdomen midway between the umbilicus and the ensiform cartilage; gradual pressure should then be exerted, penetrating more deeply with each respiratory retraction of the diaphragm until the spinal column is reached, when motions of a vibrating or trembling nature should be executed.

The splanchnic plexus is reached in the same manner, except that the straight fingers should penetrate toward the spinal column midway between the umbilicus and the symphysis.

Abdominal massage may be followed with advantage by a general vibration of the abdomen, given gently by means of an electric vibratory apparatus.

The stomach, bladder and rectum should be emptied before massage of the abdomen is begun.

ELECTRIC TREATMENT.

The use of electricity in the treatment of gastric and intestinal disorders has been highly recommended by various writers, but the general practitioner rarely avails himself of this therapeutic agent.

To Einhorn belongs the credit of bringing electrization of the stomach within the range of practical therapeutics, both by experi-

ment and by the invention of his deglutible stomach electrode. From an extensive study of the physiologic effects of direct electrization of the stomach, Einhorn draws the following conclusions:

1. Direct faradization of the stomach increases gastric secretion during the application and also for a short time afterward.

2. Direct galvanization of the stomach, with the negative pole within the organ, in most instances diminishes gastric secretion.

3. Direct faradization as well as galvanization of the stomach increases its absorbent faculty.

His conclusions as to the therapeutic value of electricity in the treatment of gastric diseases are:

1. Direct gastric electrization is a potent agent in the field of chronic (non-malignant) diseases of the stomach.

2. Direct gastrosfaradization proves to be useful in many ways in the majority of chronic diseases of the stomach.

The favorable results appear very promptly in cases of gastric dilatation not due to pyloric obstruction. Here the benefit is apparent whether there is subacidity or hyperacidity. Cases of relaxation of the cardia (eructation) and of relaxation of the pylorus (presence of bile in the stomach) have been very favorably influenced by faradization.

Gastrogalvanization is almost a sovereign means for treating severe and very obstinate gastralgias, no matter whether the pain is of nervous origin or from cicatricial ulcer.

4. Gastrogalvanization exerts a favorable influence on several affections of the heart complicated with gastralgia.

The good results obtained from electric treatment of the stomach would seem to indicate that the sensory and secretory nerves have been stimulated, although Freund made a study of the effect of the electric current on gastric secretion and found that it was absolutely negative, the only result being the production of a small amount of a mucoid secretion strongly alkaline in reaction. He concludes that food is the only stimulus which will cause the gastric glands to secrete.

Indications.—Electric treatment of the stomach is indicated in cases of atony and ptosis of the stomach and its sequelae. Favorable results may be expected in the absence of organic stenosis of the pylorus. Faradization is especially recommended in cases of gastric atony. A further indication for electric treatment is furnished by those neuroses of the stomach which, in the absence of marked objective symptoms, are to be considered as functional derangements. As examples we have paresthesias, gastralgias, pylorospasm, nervous vomiting, bulimia, and anorexia. In these cases the galvanic current is employed with good results, particularly in cases of gastralgia, hysterical vomiting, and the vomiting of pregnancy. The applications are made both intraventricularly and extraventricularly. Good results have also been secured with intra- and extraventricular faradic treatment of such conditions.

As a rule the intraventricular application of the electric current is more successful than the extraventricular. The latter is especially adapted to those cases in which the object is to exert an influence on the abdominal muscles as well as on the stomach itself. Since the normal gastric mucous membrane is not sensitive, electric treatment of the interior of the stomach is easily accomplished.

Intraventricular Electrization (*Application of Electricity to the Interior of the Stomach*).—Several apparatus are at our disposal for the application of electricity to the stomach. First of all there is the electric sound of Boas (Fig. 27). This is a stomach tube, with numerous small perforations at its lower extremity, containing in its interior a spiral of platinum that is held in place by a clump at the upper opening of the tube. Water can be introduced into or withdrawn from the stomach by means of this sound while the latter remains *in situ*.

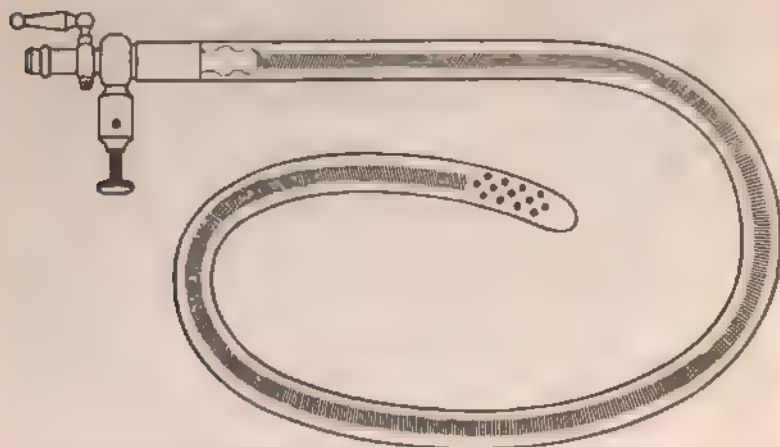


FIG. 27.—Stomach electrode. (Boas.)

Wegele makes use of an ordinary stomach tube with a glass joint at its oral end. By means of a rubber tube a funnel can be joined to it, if necessary, and the stomach either filled with or evacuated of water. A fine metal wire, ending in a button, is introduced into the stomach tube so that the button reaches to within one centimeter of the stomach end of the tube. The exact length of wire to be introduced into the tube is adjusted by a set screw (Fig. 28).

A third apparatus has been described by Einhorn (Fig. 29). A metal button within a perforated hard rubber capsule is joined by a fine transmission wire to an electric battery. The transmission wire is insulated by a thin rubber tubing. In using this apparatus the patient swallows the hard rubber capsule with a little water.

McKewood has modified Einhorn's gastric electrode by making the following changes: The capsule is reduced in size to the dimensions of an ordinary five-grain gelatin capsule. To the metal button

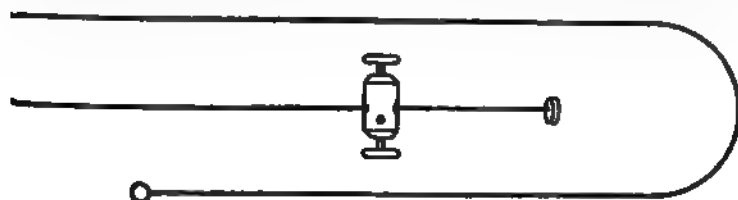


FIG. 28.—Stomach electrode. (Wegels.)

within the capsule is attached a spiral of flat steel, the flexibility of which corresponds to that of an ordinary stomach tube. This spiral is covered by thin rubber tubing, and is tipped with a binding pin for connection with the battery.

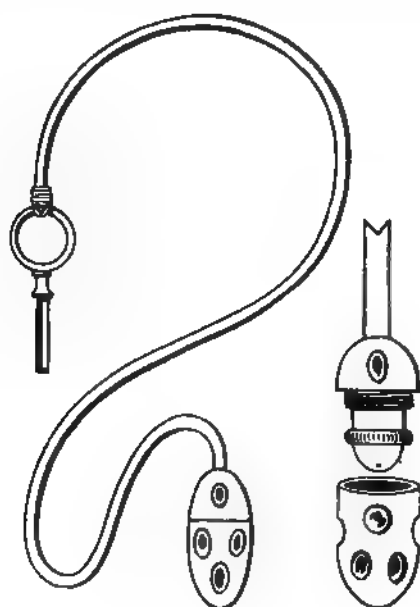


FIG. 29.—Intragastric electrode. (Einhorn.)

which an electrode can be easily introduced into the stomach without discomfort. The small size of the capsule allows of its easy passage, while the spiral attachment is sufficiently resistant to enable the operator to push the capsule along, just as a stomach tube is introduced.

Stockton has devised an instrument which is a combined stomach tube and electrode (Fig. 30). An ordinary soft rubber stomach tube, 28 inches long, is coupled by means of a ground steel joint to three feet of rubber tubing, terminating in the ordinary funnel. Through this the stomach is emptied in the usual way. Then the rubber tubing is disconnected at the coupling without removing the stomach tube itself from the stomach. There is now introduced through the stomach tube, *in situ*, a spiral electrode, which, when in place, completely closes the proximal opening of the tube by a ground steel plug, the distal extremity of the wire being at the upper of the two fenestræ at the lower end of the tube. This arrangement prevents the metal point from touching the mucous membrane of the stomach.

Before the introduction of the electric sound the patient drinks a large tumblerful of lukewarm water, or the water may be introduced by means of the tube. The fluid distributes the current to

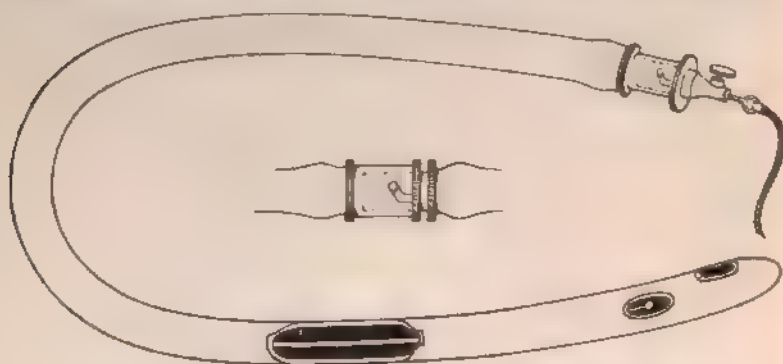


FIG. 30.—Combined stomach tube and electrode. (Stockton.)

the gastric wall. For the purpose of faradization a large plate electrode is placed either on the epigastric region or on the back, to the left of the seventh dorsal vertebra. Weak currents are employed at first, the current being gradually increased to such a force that the patient is just able to bear it. Rather forcible currents are permissible. The duration of the seance is about ten minutes.

The negative electrode is introduced into the stomach for the purpose of galvanization. A broad plate electrode is applied in the same manner as when faradizing, the location being altered if necessary; the current is begun slowly and carried up to the strength of 15 to 20 milliamperes, and is then slowly diminished. The duration of the treatment is from eight to ten minutes.

Galvanofaradization is likewise applicable. Intraventricular faradization is especially recommended in atony, relaxation of the pylorus, and paresis of the cardia from disturbances in the cerebral nerve centers or from neurasthenia. Internal galvaniza-

tion is especially worthy of application in gastralgias, chronic hypersecretion, and derangements of the autonomic nervous system. The internal electric treatment of the stomach is the sovereign remedy for nervous vomiting.

Extraventricular Electrization.—Two large rectangular plate electrodes are to be employed for this purpose. One of them, well moistened, is to be applied to the region of the stomach, the other to the back. The gastric electrode is put on firmly and pressed deeply, making the distance between the two plates as small as possible. Another arrangement of electrodes is as follows: Of two large curved plates, the larger one (300 millimeters square) is applied from the front of the abdomen to the spinal column, and the other in a similar way on the opposite side. The distance between the edges of the two electrodes must be at least one or two centimeters.

While faradizing, weak currents are used to begin with; these are gradually increased, as in intraventricular faradization, to the limit of the patient's tolerance. In sensitive persons treatment may be interrupted by a short pause every half-minute.

An electric roller cylinder may be employed instead of the anterior electrode; this is rolled to and fro in the region of the stomach without interruption, and thus effects an even electric massage of the stomach. The electric brush may likewise be used anteriorly.

While galvanizing, the current is gradually increased to 15 to 20 milliamperes, it being a matter of indifference whether the electrode is situated in front or behind. The duration of the treatment is five minutes.

High-frequency currents have been extensively used in the treatment of diseases of the digestive organs. A million volts can be made to permeate the body by a course of autocondensation. As a result, metabolism is increased and muscular contractions stimulated, while neural and glandular excitement is quieted. The effects upon the intestine seem to be more gratifying than those upon the stomach. This form of electricity is employed in gastric atony and gastralgia, but more often in intestinal neuroses, membranous colitis, and atonic and spastic constipation. It is also used in the treatment of anal fissure (see page 861).

The alternating faradic (sinusoidal) current in which the potential rises gradually from zero to a maximum, and then gradually returns to zero or to a minimum, gives splendid results.

Static electricity in the treatment of diseases of the digestive organs is disappointing.

CHAPTER XI.

TREATMENT OF DISEASES OF THE INTESTINE THROUGH THE RECTUM.

RECTAL therapeutics plays an important rôle in the treatment of diseases of the intestine. This class of therapeutic procedures should fulfil one of two indications: (1) To empty the bowel by stimulating peristalsis; that is, to act purgatively. (2) To act upon the intestine in such a manner that inflammatory processes of the diseased intestinal mucous membrane are improved by direct local treatment, retarding peristalsis and checking diarrhea; in other words, securing a constipating effect.

The first-mentioned indication is attained by the so-called cleansing enemata. These are made use of in all acute and chronic intestinal stagnations, both primary and secondary. Cleansing enemata are most extensively employed in chronic constipation. For this purpose they are more valuable than purgatives. When it is impossible to attain a satisfactory result by dietetic means, enemata are first to be used, and only in case of their failure should we resort to purgatives.

CLEANSING ENEMATA.

The technic of cleansing enemata is as follows: A soft rectal tube, made of the same kind of rubber as that used for stomach tubes, is employed. This rectal tube may be closed at its lower end, with one or two lateral apertures, or it may have one opening at the extreme end and one lateral opening. The edges of these openings must be smoothed and rounded, in order to guard against any injury to the mucous membrane of the rectum. The caliber of the rectal tube should be as large as possible, especially if it be desired that the outflow of the water take place through the same tube. The lower half of an ordinary rubber stomach tube (Fig. 31), having a closed point and two lateral openings, is best adapted for use as a rectal tube. This instrument rarely becomes stopped up. The end of the tube should be well oiled and introduced into the rectum of the patient for a distance of about ten to fifteen centimeters (3 to 5 inches) with careful pressure. The tube should never be forced farther than can be accomplished without a feeling of resistance. It is usually easy to pass the tube high up while fluid is flowing into the rectum. Hard tubes made of wood, vulcan-

ized rubber, or glass, as substitutes for the soft rubber rectal tubes, are to be used with great caution, because they may easily injure the mucous membrane even when skillfully handled. After the introduction of the soft rubber rectal tube, the latter is joined to a piece of wide glass tubing and this is connected with a rubber tube which is attached to an irrigator of glass having a capacity of about 1500 Cc. (3 pints). This apparatus corresponds exactly to that used in lavage of the stomach. The irrigator is graduated, and constructed in such a manner that it may be suspended from a hook. The diameter of the outflow opening should be as wide as possible. The tube used for making connection should be of the best thick black rubber. One end of the connecting glass tubing should be somewhat narrower than the other, that it may be easily introduced into the rectal tube. A large glass funnel may be used instead of the irrigator, though the latter has the advantage that it can be placed on the floor and the outflow of the water can then be easily watched. The pressure of the inflowing water can be easily regulated by elevating or lowering the irrigator or funnel. This is of great importance, especially in patients with a very sensitive intestine and in those suffering with tenesmus. The employment of

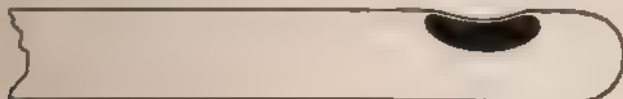


FIG. 31. - Rectal tube, showing solid end and elongated lateral opening.

piston syringes and rubber-bulb syringes is not to be recommended; they are sometimes connected with the rectal tube after the latter is introduced. It is only with great difficulty that intra-intestinal pressure can be regulated in the use of these syringes, and over-distention of the gut may result, causing pains and tenesmus. Another practical drawback is the fact that the syringes are not capable of holding a sufficient quantity of water.

It is useless to insert the rectal tube farther than the distance mentioned above (10 to 15 centimeters). The so-called "high colonic irrigations," which require the introduction of the rectal tube high up, have been found to be practically impossible. Roentgen fluoroscopy shows that a soft rubber tube cannot be made to go any higher than six or seven inches into the rectal ampulla without bending or coiling on itself. Only in rare cases can it be made to penetrate into the colon. To completely fill the colon with liquid it is not necessary to insert the tube very high. The result desired is accomplished much better by allowing the patient to assume a suitable posture—either the left lateral or the knee-chest. The intra-abdominal pressure is thus diminished and the fluid is sucked up, as it were. By this means large quantities of

liquid can be made to ascend, under low pressure, through the colon into the cecum. The ileocecal valve can rarely be passed by the fluid. To completely fill the entire large intestine, six liters of liquid are required.

With the apparatus mentioned above, the patient is enabled to administer these enemata himself. In such an event the rectal tube had better be joined to the irrigator tube (Fig. 16) before being introduced into the rectum. The irrigator being suspended at a moderate height, and the fluid allowed to flow as far as the end of the tube in order to expel all the air that may be present, the tube is then shut off by means of a large tube-clamp. The patient assumes the left lateral position, with the pelvis slightly elevated. The rectal tube is inserted, and the flow and pressure of water are regulated at will by manipulation of the clamp.

The quantity of water to be introduced should not be too large. About 300 Cc. (10 ounces) of liquid, flowing in under low pressure, is sufficient for cases of chronic constipation in which the patient is obliged to resort to enemata in order to "move" the bowels. Regular and long-continued employment of large-sized enemata, one liter or more, is actually harmful, for a marked atony and overdistention of the rectum may result, causing a weakness of the muscular tissues. The chief point is that the small enemata, of 300 Cc. (10 ounces), should be retained as long as possible—for hours, or if possible during the entire night. For this reason it is well to have the injections made at night, evacuation taking place the following morning. The purgative effect of these enemata is brought about in the following manner: The peristalsis is mechanically stimulated by the inflowing water, the slight distention of the intestinal wall, and the irritation from the tube in the rectum. The degree of increase in the peristalsis is dependent on the temperature of the liquid; cold irrigations (65° to 70° F.) act more powerfully than warmer ones. Then there is the softening effect of the water on the firm fecal mass to be taken into consideration. This effect is only obtainable when the enema is retained for a long time. Ordinary water of different temperatures is frequently efficacious, but it is sometimes advisable to add to the water various substances, such as chamomile tea, soap, oil, glycerin, sugar, honey, vinegar, soda, common salt, molasses, castor oil, or oil of turpentine. Soap has the most energetic action in dissolving fecal matter, and oil stands next. Lime-water has also been recommended.

Glycerin Enemata.—Small glycerin enemata (about 5 to 15 Cc.), slowly injected into the rectum, by abstracting water from the tissues reflexly stimulate peristalsis and frequently cause evacuation within a few minutes. This peristaltic stimulation extends over the lowest portion of the gut only; the use of glycerin is rational and effective, therefore, only for emptying the rectum.

Glycerin in suppository form acts in the same manner. In nervous patients the use of glycerin occasionally gives rise to nausea and vomiting.

Oil Enemata.—Fleiner's oil enemata are extensively used in the treatment of chronic atonic and spastic constipation. Fleiner recommends for this class of cases one injection daily of 250 to 500 Cc. ($\frac{1}{2}$ to 1 pint) of the purest olive or sesame oil. The oil is to be retained in the bowel for a considerable time; it is best to retain it overnight if possible. Should discomfort during the night (meteorism, pressure) result, as may occasionally happen, the time of administration should be changed, the enema being given at six or seven o'clock in the morning while the patient is in bed; the oil is then to be retained for three or four hours, thus producing the same laxative effect as if it had been administered at night.

These injections should be continued for several months, at first daily, later every other day, and subsequently twice a week. The results are so good that in many cases of chronic constipation actual recovery is brought about without any other treatment. The oil, by partially breaking up into fatty acids, stimulates peristalsis. When there is spastic constipation the oil has a soothing effect on the tense muscular tissue; and in atonic constipation it strengthens the muscular tonus. Besides it lubricates the gut, softens the fecal agglomerations, and forms a protective layer upon the inflamed portions of the mucous membrane (see page 671).

As a rule no discomfort is caused by the "oil cure," and the patients are at the time hardly aware of the fact that the oil is being introduced. It has not been ascertained definitely whether the oil passes beyond the ileocecal valve in all cases, but some patients experience the taste of oil after receiving a number of enemata. The only inconvenience caused by the oil enemata is the impossibility of avoiding the soiling of the bed and the bed-clothes. The patient must remain in bed for at least an hour after the injection, without indulging in much conversation, coughing or laughing. Should there be no spontaneous action of the bowels in the morning, a small lukewarm sodium-chlorid water enema should be given.

The oil may be made to flow through the apparatus previously described, the irrigator being held high. Because of the viscosity of the oil, the pressure may have to be somewhat stronger than usual. A piston syringe of glass, hard rubber, or metal, holding 300 Cc. (10 ounces), may be used; the nozzle is to be attached to a soft rubber rectal tube. The syringe should be elevated during the injection, and considerable pressure may be made upon it. Zweig (Fig. 32) has constructed an oil enemator after the manner of a squirting bottle. The oil is placed in an Erlenmeyer flask (a) which is closed by a doubly perforated rubber stopper (b). Through

one of the openings in the stopper a glass tube (*c*) passes to the bottom of the flask; the outer extremity is then joined to the rectal tube (*f*) by a piece of rubber tubing (*d*) with glass connection (*e*). Through the other opening in the rubber stopper a short tube is inserted, care being taken that it does not touch the level of the oil. As in the squirting bottle, air is forced into the flask by means of a double rubber-bulb apparatus (*g*) under gentle but uniform pressure.

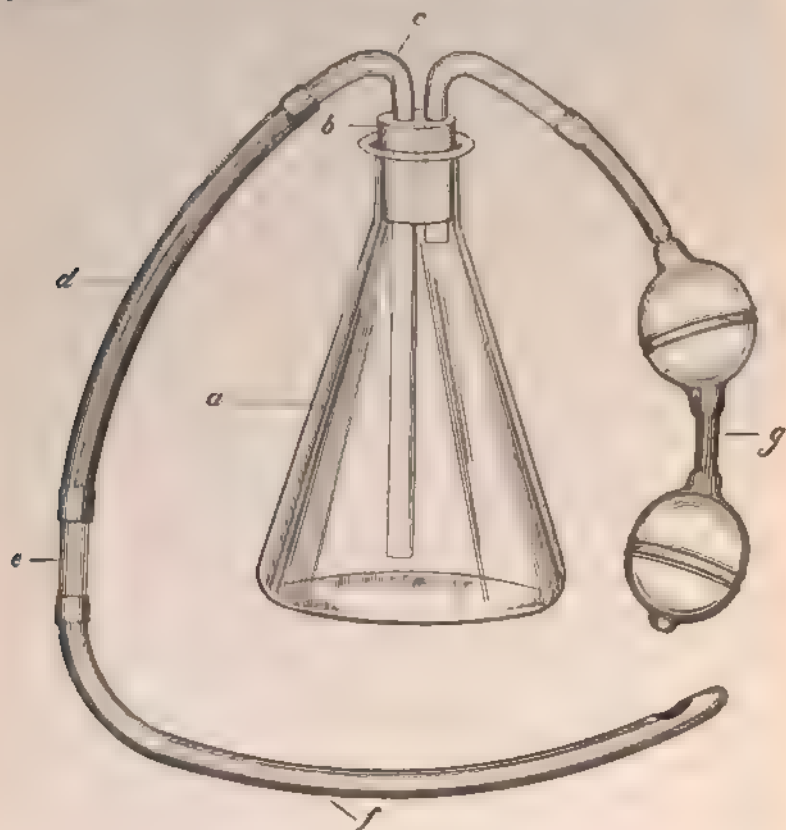


FIG. 32—Oil eucuator (Zweig). *a*, Erlenmeyer flask; *b*, doubly perforated rubber stopper; *c*, glass tube; *d*, rubber tube; *e*, glass connection; *f*, rectal tube; *g*, double rubber bulb.

An apparatus for the injection of oil devised by Dudley D. Roberts consists of a curved rectal tube of hard rubber, ending in an olive-shaped nozzle, and attached, by means of a soft rubber tube of convenient length, to a ten-ounce Politzer bag (Fig. 33). The rectal tube is made with such a curve that it is readily passed from the front of the body to the anal opening; here by a slight traction movement it enters the anus in the proper direction, pointing toward

the umbilicus. This new form of tube is much less awkwardly introduced than by reaching around behind the buttocks. The Politzer bag is fitted with a stopcock having a small hole in one side, which permits the bag to fill with air when the cock is closed. The method of taking the injection is simple: The bag is allowed to fill itself, and the oil is then warmed by placing the bag in warm water. The patient lies on his back, with hips somewhat elevated, and introduces the rectal tube with the soft rubber tubing attached. The bag is then attached, the stopcock turned, and the oil slowly

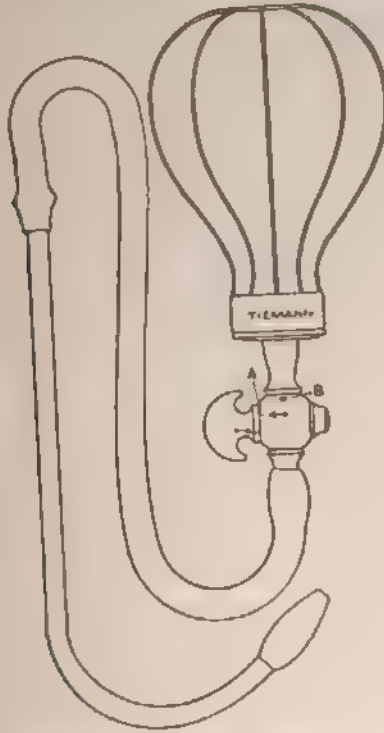


FIG. 33.—Oil enemator. (Roberta.)

forced into the rectum. The bag, being emptied, is closed by reversing the stopcock; dilated by the air which thus gains entrance; and compressed again to force out what oil remains in the tubing. By shutting off the stopcock we prevent the return of any oil into the tube and obviate the danger of soiling clothing and bedding.

Paraffin Enemata.—Lipowski has quite recently advocated the paraffin mixture instead of oil. The hardness of the stools in chronic constipation being, as he says, due to the fact that much more water is absorbed by the mucous membrane of the sigmoid

flexure and the rectum than in health, it is desirable to prevent this loss of water if possible. This he attempts to do by the introduction of agents into the rectum which retard absorption. Paraffin, according to Lipowski, has this effect. He makes use of a preparation that has a melting point of about 100° F., consisting of solid paraffin (melting point 160° F.) and liquid petrolatum in the proportion of one of the former to eight of the latter. The mixture is heated, either in a water-bath or over a flame, until it becomes all liquid. Of this, 200 Cc. (7 ounces) is slowly injected through a soft rubber rectal tube. Several advantages are claimed for the paraffin injections, among others that they do not necessitate lying down for hours after the injection as oil enemata do, and the paraffin mass spreads over the surface of the mucous membrane in the manner of an ointment very soon after the injection. The paraffin is absolutely non-irritating and it does not decompose, as ordinary oils do. In the paraffin treatment large volumes of flatus are not formed, as may happen in the oil treatment, and the clothing and bedding are not soiled by malodorous masses. The purgative effect of paraffin is said to be much more prompt than that of oil. Paraffin is very well adapted for continued use, because of its absolutely non-irritating character. The patients are able to apply the injections themselves. Lipowski has reported permanently good results from this method of treatment.

Carbon Dioxid.—Adolf Schmidt, taking into consideration the fact that gases are a physiologic stimulant to peristaltic movement, has proposed to employ as enemata liquids containing carbon dioxid, such as selters water and carbonated oil of sesame. These enemata may be introduced by allowing the fluids to pass directly into the rectum from the bottle—a rectal tube attached to a rubber tube being connected with the neck of the bottle. By turning, shaking and inverting the bottle, carbon dioxid is developed in large volume, forcing the contents of the bottle into the rectum. The ordinary selters-water bottle as found in the shops can be used; a rectal tube can be easily attached to the faucet (Fig. 34). These enemata should be retained as long as possible, although it is not feasible to hold them as long as the non-gaseous fluids. The carbon dioxid has a refreshing influence on the mucous membrane of the intestine and stimulates both tonus and peristalsis.

Bile Enemata.—Bensaude reports good results in the treatment of constipation by means of bile enemata. Of interest is the fact that this is one of the oldest measures known in medicine, for 1300 years before Christ a mixture of beef bile and milk, in the proportion of one-third bile and two-thirds milk, was used as an enema for constipation by the Egyptians. Bile is capable of exciting contraction of the colon as well as of the small intestine. A single dose of 10 Gm. (2½ drachms) of bile causes colicky contractions. The dry extract dissolves freely in water. A half-liter (1 pint) of the solution is

allowed to enter the rectum by gravity; and in five to ten minutes an evacuation of the bowel occurs.

Cathartics.—Attempts have also been made to secure a purgative effect by the introduction of small enemata containing cathartics. The active principles of various purgatives have been tried—aloïn, colocynthin, cathartin, and citrullin. This method, however, is as yet only in the experimental stage.

All the above-mentioned measures refer principally to the overcoming of chronic constipation. The administration of water enemata with or without some adjuvant must also be considered in the treatment of acute constipation; in acute and very persistent constipation with organic cause, as much as 1½ liters (3 pints) may be introduced, to be retained for a short time only, and the injection repeated if necessary.

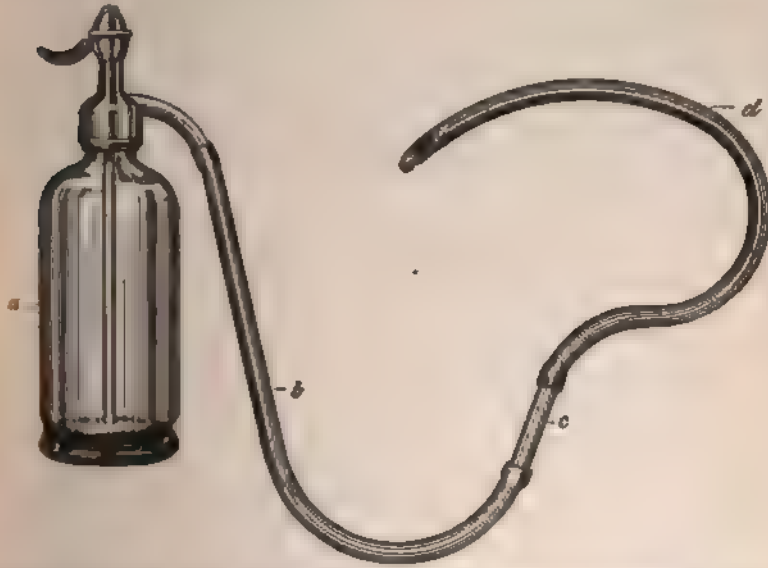


FIG. 34.—Carbon dioxide bottle with soft rubber rectal tube attached. *a*, siphon bottle; *b*, rubber tube; *c*, glass connection; *d*, soft rubber catheter.

In stenosis of the large intestine the cause of the constipation is usually the accumulation of coarse remnants of food above the stricture. By administering small or large enemata, rapidly repeated by alternately elevating and lowering the irrigator, the obstruction may in many cases be removed. When the occlusion is caused by gallstones, fecal concretions, or swallowed foreign substances, it is often possible to soften and remove the obstructive masses by frequent and dextrous rectal irrigations. Boas warmly recommends chloroform-water irrigations (10 : 200) in cases of gallstone ileus for the purpose of partially dissolving the concre-

ments. These irrigations, with large quantities of fluid, sometimes require general anesthesia, particularly when it is desired to overcome the resistance at the ileocecal valve.

To relieve intussusceptions, rectal irrigations are very serviceable. Nothnagel used 5 to 8 per cent. sodium chlorid solutions in these particular cases. To remove the gases found in meteorism, large enemata are likewise to be employed and rapidly evacuated; the water returning under pressure is capable of carrying away with it large quantities of gas. It may be mentioned that Curschmann recommended the insufflation of air into the rectum in cases of constipation due to stenosis caused by tumor or intussusception. This procedure, however, is not without danger, because of the liability of tearing or causing perforation, particularly in ulcerative and gangrenous processes. The insufflation of air is accomplished by means of a double rubber-bulb apparatus attached to a rectal tube.

Intestinal Douche.—The intestinal douche is employed to stimulate peristalsis and to strengthen the muscles of the lower section of the bowel. The douche is applied with a rectal tube similar to Rosenheim's gastric douche, which is provided with numerous small openings at its lower end through which the fluid is expelled in jets (Fig. 35). The liquids used are either cold water, or hot and



FIG. 35.—Perforated tube for rectal douche.

cold water alternately, or water containing carbon dioxid. The rectum should be empty at the time; a cleansing enema will empty it before using the douche.

MECHANICAL TREATMENT.

Swedish Manipulation.—Certain manual methods, apart from the use of enemata, are at our disposal for bringing about evacuation of the bowel. Akerhielm recently recommended Swedish manipulation in cases of very obstinate chronic constipation. The patient is placed on his left side upon a low couch, with the knees well flexed. The attending physician stands behind, and exerts a light counterpressure with the left hand on the patient's upper hip; his right index finger, covered with a well-oiled rubber glove or a thin finger cot, is then inserted into the rectum. The palm of the hand is turned outward, away from the genital region of the patient, and the finger is slightly bent; both anal sphincters are then carefully passed until the ampulla is reached. Normally the mass of fecal matter pressing downward during defecation mechanically stimulates the walls of the rectum, and this stimulus,

transmitted by fibers from the hemorrhoidal plexus to the anospinal center, reflexly induces evacuation. Akerhielm endeavors to imitate or replace this impetus, which is absent in chronic constipation, by executing frictional movements with the inserted finger along the walls of the rectum. All parts belonging to the genital apparatus are most carefully avoided. The manipulation should be directed toward the lateral soft parts of the ampulla, where the hemorrhoidal plexus is embedded. The stimulation must not be applied with too much vigor, although the sensitiveness in the interior of the rectum is not very pronounced; it must always be borne in mind that these tissues are very tender and markedly vascular. The stimulations are to be made slowly, and the whole procedure should not continue longer than two or three minutes. Akerhielm does not give any purgatives or enemata. He succeeds in effecting a bowel movement in his patients, even a copious one, by means of a single treatment. The feces become moist, the mucous membrane lubricated, and an evacuation takes place within ten hours after the manipulation. The cure is said to take from four to six weeks, the manipulation being done once daily. In the course of six months to a year a short after-cure series of manipulations should be undertaken.



FIG. 36.—Apparatus for rectal massage. a, inflated; b, deflated.

Rectal Massage.—Hirschman has devised a method of rectal massage with a pneumatic dilator mounted on a bougie, which is quite simple. The instrument consists of an ordinary Wales bougie, supplied with an inflating bulb, and covered at its distal end with a thin rubber bag (Fig. 36). The rubber bag, well lubricated, is inserted into the rectum with the patient in the Sims position. It is passed inward, backward, and upward, following the sacral curve to the entrance into the sigmoid; it is then slowly inflated for about ten to fifteen seconds. The bag should be allowed to empty as soon as its distention causes a feeling of fulness or cramp. This inflation and deflation, four to six times a minute, should be kept up for five to ten minutes; then the bag is inflated to the point of tolerance, the openings in the bougie stopped with the thumb and forefinger, and the apparatus withdrawn with a toward-from movement, thus massaging the sphincter and rectum in its exit. With instructions to the patient regarding regular hours for

defecation, the proper diet and exercise, and rectal massage persisted in until a daily stool is produced, Hirschman has found the treatment successful in a vast majority of his cases. He has had less than 5 per cent. of failures.

Rectal Tampons.—MacMillan has used rectal tampons in a large number of cases of chronic constipation with most gratifying results. A tampon placed above the rectal valves, and allowed to remain for three or four hours, will usually ensure a bowel movement within twenty-four hours, and in most instances another movement before the expiration of another twenty-four hours. The tampons are composed of cotton, lamb's wool, or gauze. They are easily inserted through a proctoscope (Fig. 37), and cause the patient no pain or inconvenience; he may leave the office with directions to withdraw the tampon in three or four hours. In the majority of cases a tampon every second day for two weeks is sufficient to effect a cure. The tampon is solid and resembles sufficiently in consistency the normal feces in the rectum or sigmoid. It does not cause any chemical irritation. Any desired degree of distention may be obtained by increasing the size of the tampon. The tampon may be retained for a considerable length of time; and to this close resemblance to the natural stimuli MacMillan believes much of its therapeutic value is due.



FIG. 37.—Kelly's proctoscope.

Rectal Electrodes.—The electric current applied in the rectum is capable of stimulating peristalsis. The apparatus used for this purpose is identical in construction with the electric stomach tube of Boas (Fig. 38), and admits of both inflow and outflow of water. The electrode being introduced into the rectum, 100 to 200 Cc. (3 to 7 ounces) of lukewarm sodium-chlorid solution is injected before turning on the current. The other electrode, consisting of a broad plate or a roller, is placed upon the abdomen and moved along in the direction of the large intestine. Either the faradic or the galvanic current may be used. The faradic current may be made strong enough to produce distinct muscular contractions.

The patient should not experience more than a distinct, slightly painful prickling sensation. With the galvanic current, twenty to

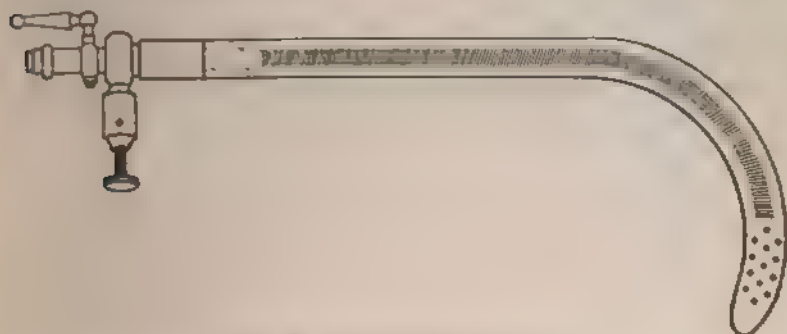


FIG. 38.—Rectal electrode. (Boas.)

thirty milliamperes are sufficient. After the treatment has been concluded, the water is allowed to flow out directly through the electrode. Generally speaking, electricity should be applied intra-

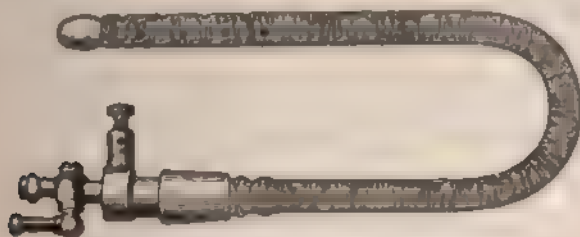


FIG. 39.—Rectal electrode. (Zweig.)

rectally only in cases in which the other methods of treatment do not accomplish the purpose. The sinusoidal current is the one generally used.

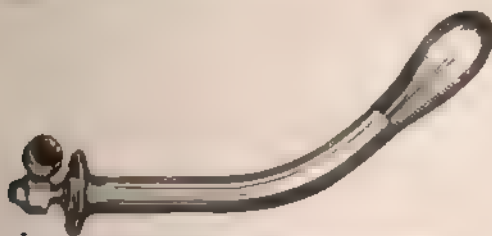


FIG. 40.—Curved rectal electrode.

Zweig also has constructed a rectal electrode (Fig. 39), with a metal screw head, which allows the inflow and outflow of water.

Figs. 40 and 41 show two simple electrodes with metal buttons, without provision for the inflow of water. The faradic current

only should be used with this form of electrode, as the mucous membrane might be cauterized from the galvanic current.



FIG. 43.—Straight rectal electrode.

An enema of water to cleanse the rectum is not absolutely necessary before faradization. The proper duration of intrarectal faradization is five to ten minutes. The current is at first applied very weak, then increased, and finally very gradually and slowly decreased. Usually the galvanic current is employed in irritable conditions of the gut, the faradic or sinusoidal current being reserved for relaxed conditions.

TREATMENT OF INTESTINAL IRRITATION.

Irrigation of the Intestine.—Irrigation of the intestine is the best procedure for fulfilling the second indication in rectal intestinal treatment; it effects a cure of the morbid processes by direct action on the intestinal mucous membrane, thus arresting the diarrhea. The purpose of irrigation is to remove noxious material, such as blood, mucus, pus, putrefying and fermenting material, to neutralize the harmful effects of these substances on the intestinal mucous membrane, and to produce a direct therapeutic effect.

Intestinal irrigations for the removal of noxious materials are performed with the apparatus mentioned on page 199 (Fig. 16). The patient places himself in the left lateral position, and the irrigating fluid is allowed to enter the rectum under any desired pressure, regulated by elevation or lowering of the irrigator. The liquid is allowed to escape immediately, or after it has been retained for some time. When it is desired to irrigate the entire large intestine, greater quantities of irrigating fluid, 1 to 1½ liters (2 to 3 pints), are required, which should be allowed to flow in very slowly while the pelvis is somewhat elevated. By placing the irrigator on the floor the return of the greater part of the fluid is effected, just as in the case of gastric lavage, and the quantity and quality of the masses removed can be satisfactorily observed in the irrigator (which is of glass). These irrigations may be frequently repeated, always with gentle pressure to avoid overstretching the intestine. Rectal tubes and joint pieces of rather large caliber should be used in order to avoid obstruction by the fecal masses. At times, before allowing the fluid to escape, the rectal tube may be entirely removed and the liquid evacuated directly into a bed-pan. It is obvious that these irrigations must be made most carefully to avoid excess-

sive pressure; the sensations of the patient as regards pain or tenesmus afford a reliable guide. Should the disease be situated in the descending colon, in the sigmoid flexure, or in the rectum, the amount of fluid used need not be large.

The apparatus mentioned on page 199 (Fig. 16) is sufficient for all purposes of irrigation. Zweig has constructed a special irrigating tube (Fig. 42), consisting of a strong piece of glass tubing rounded at the end and with two oval openings two or three centimeters from the end, one on each side. The lumen of the glass tube is divided longitudinally by a diaphragm into two non-communicat-

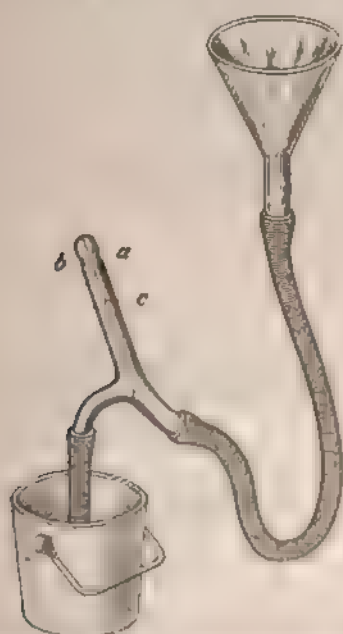


FIG. 42—Irrigation tube (Zweig) *a*, small opening in glass tube leading to funnel *b* opening through which water flows out of rectum, *c*, diaphragm dividing glass tube into two halves.

ing halves. One of these halves is connected with a rubber tube attached to a funnel, and serves for the inlet of the water, while the other half is attached to a shorter rubber tube leading the outflowing water into a vessel. The irrigation is made in the following manner: The well oiled glass tube is inserted into the anus of the patient, who is lying on his left side. Water being allowed to flow from the irrigator, the air present in the apparatus escapes with a noise through *a* into the rectum and from there through *b* into the outflow tube. Now follows the irrigating water, which washes and rinses the rectum and leaves it by *b*. Water is continuously or at short intervals poured into the elevated funnel

or irrigator until the rectum has been thoroughly washed. When it is desired to irrigate higher up, the outflow rubber tube is clamped off; the water is then unable to escape so rapidly and must first pass into the higher sections of the intestine.

Rosenberg recently constructed an irrigation apparatus which is all metal (Fig. 43). It is somewhat longer than the normal rectum. When inserted, its upper cupola lies in the inferior end of the sigmoid flexure and thus prevents the irrigating fluid from passing higher up. In the axis of the apparatus is situated an irrigation tube provided with numerous apertures (*b*) through which the fluid enters the rectum. The fluid escapes by the outflow tube (*c*). This irrigation tube (*b*) is easily removable from the apparatus and may be replaced by the tube *d* with which direct application of medicinal substances can be made.

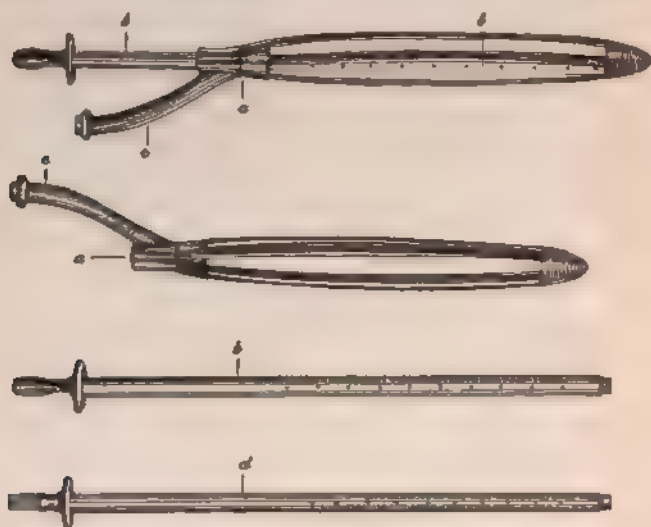


FIG. 43 —Irrigation apparatus. (Rosenberg.) *a*, outside metal covering; *b*, irrigating tube with apertures; *c*, outflow tube; *d*, for direct application of medicaments.

Wolbarst has devised an improved rectal irrigation tube of great value (Figs. 44 and 45). The tip of the instrument is of soft, pliable rubber, which bends easily on contact with the rectal wall, and the flow of liquid into the rectum is through numerous small openings, thus providing a fountain spray instead of a single or double jet. There are two tubes, one inside the other. The water enters through the small tube (*a*) and fills up the soft rubber pouch (*b*), through which it enters the rectum. Escape from the rectum is only possible through a large opening (*d*) in the larger tube (*c*). The external sphincter (at *f*) prevents any outflow at the anus.

The instrument is made of brass tubing, nickel plated, with a

soft rubber tip; the total length is seven inches, including the tip. The diameter is equivalent to 38 of the French scale. The soft rubber tip projects $1\frac{1}{2}$ inches beyond the end of the large tube; it is slipped over the bulbous end of a short metal tube or ring, the



FIG. 44.—Rectal irrigation tube. (Wolbarst.)

other end of which is provided with a male thread which screws into the end of the large tube (c), and is thus securely wedged in place. It is easy, therefore, to unscrew the rubber tip and thoroughly clean it and the metal tubes at will.

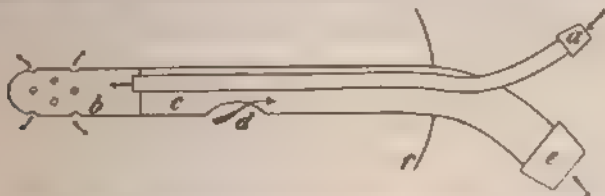


FIG. 45.—Rectal irrigation tube, sectional view. (Wolbarst.)

Antiseptics.—Warm water, normal saline, or weak filtered chamomile tea is used for irrigation. After the gut has been cleansed, antiseptic or mucus-solvent substances may be used for irrigation, to act directly upon the mucous membrane. The antiseptic drugs are boric acid (one teaspoonful to a liter of water), salicylic acid (1:1000), salicylate of sodium (1:1000), weak solutions of permanganate of potassium (1:10,000), lysol ($1\frac{1}{2}$:1000), thymol (one teaspoonful of a 1-per-cent, alcoholic solution to one liter of water), ichthyol, benzoate of sodium, naphthol (1:1000), quinine hydrochlorid (1:1000), and occasionally iodoform emulsion. For dissolving mucus we may use the thermal waters of Carlsbad, or Carlsbad salt in solution, in quantities of about 200 Cc. (7 ounces). Lime-water, and weak solutions of sodium carbonate, sodium acetate or boric acid, are mucus-solvents. Application of these solutions should be frequently made, always just after the bowel has been cleansed. Each treatment should be continued for five to ten minutes. Such cleansing and disinfecting irrigations are applicable in cases of chronic colitis, especially when associated with ulcerations, and in stenosis, ulcers, and ulcerating tumors associated with profuse secretion of pus, blood, and serum. In the latter case only small quantities are to be employed at one time, and then very carefully.

Sedatives.—Sedative irrigations may be employed when there is much irritation, pain, and tenesmus—which condition is found in dysentery and other serious inflammatory processes in the large intestine and the rectum. Here, as a rule, the basis of the irrigation is a weak decoction of starch, a thin mucilage of gum arabic, or a decoction of chamomile tea or peppermint, to which is added 0.5 to 2 Cc. (10 to 30 drops) of tincture of opium, 0.02 to 0.05 Gm. ($\frac{1}{2}$ to 1 grain) extract of belladonna, or 2 to 4 Gm. (30 to 60 grains) of chloral hydrate. These enemata are to be retained for a considerable time. Opium and extract of belladonna act quite well in such cases when applied in the form of suppositories.

Astringents.—Astringents may also be applied to the intestinal mucous membrane by way of the rectum, astringent medicaments being added to the irrigating fluids. Silver nitrate was formerly used extensively (1:500 to 1:1000), but has been very properly abandoned because of the excessive irritation that so frequently followed its use. Tannic acid (one teaspoonful to a pint of water) has been used more extensively, but it, too, may prove very irritating, and is not now used so often as formerly. Aluminum acetotartrate (one teaspoonful to the pint of water) seems to be less irritating. Some insoluble astringents are much milder, *e. g.*, bismuth subnitrate and bismuth subgallate. Suspensions of these drugs are quite effective, especially when made with starch or gum arabic. If they are allowed to remain for some minutes in the intestine, the patient being appropriately placed, a portion at least of the drug is precipitated on the mucosa and thus enabled to exert its curative action. Such injections are particularly adapted to the treatment of ulcerative and severe catarrhal processes.

The best results in all catarrhal conditions of the mucous membrane of the rectum, sigmoid or colon are obtained by the use of krameria. The solutions as ordinarily found in the shops are useless. The preparation which is recommended by Tuttle and which I have found extremely valuable is prepared as follows:

"Macerate one pound of bark of krameria in a long percolating tube for twenty-four hours. After this a mixture of 20 per cent. glycerin and 80 per cent. water is allowed to percolate through it. The percolate should be constantly stirred, and filtered through the bark a second time. The filtrate is then evaporated down to one pound, thus obtaining an aqueous fluid extract containing minim for grain all the therapeutic properties of the bark. The preparation should be kept in a dark place and not exposed to the air."

This aqueous solution of krameria mixes freely with water and can be diluted for irrigation to a strength of from 2 to 20 per cent. For local applications it can be applied full strength. It has an astringent and anodyne effect and can be applied to the tenderest rectum without irritation.

Natural Mineral Waters.—The thermal waters of Carlsbad, Wiesbaden, Ems and Neuenahr are likewise worthy of frequent trial, in small quantities, 100 to 200 C.c. (3 to 7 ounces), especially for chronic catarrh of the large intestine. The effects of these waters are dependent partly on their mucus-solvent action, but they also have a directly beneficial effect on the intestinal mucous membrane in catarrhal conditions. They may also be employed in the treatment of ulcerative processes.

Dry Treatment.—Dry local treatment of catarrhal and ulcerative colitic processes has been highly recommended of late. This treatment must be administered with the assistance of the sigmoidoscope, and may therefore be applied only as far as the sigmoid flexure. Many instruments are available for this purpose; the one usually employed is the pneumatic sigmoidoscope (Fig. 46). A

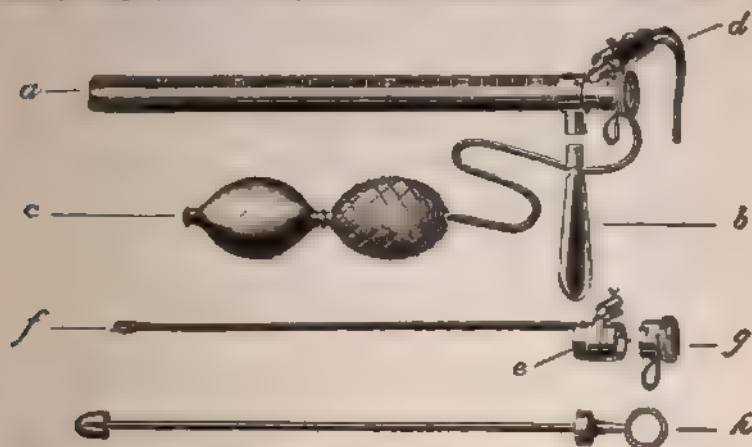


FIG. 46.—Pneumatic sigmoidoscope (Strauss) a, metal cylindrical tube, b, handle c, double rubber bulb, d, electric wires, e, staff carrying light, f, lamp, g, glass end-piece, h, obturator.

metal cylindrical tube (a), 35 centimeters (12 inches) long, with a handle (b), is attached to a double rubber bulb. A metal obturator (h) closes the inner end of the speculum before it is introduced into the rectum. After the instrument has passed through the anal canal, the obturator (h) is removed and a metal staff (e) carrying an electric lamp (f) is introduced in its place. An electric wire (d) connects the lamp with a storage battery. By attaching the glass end-piece (g) and gradually inflating the rectum with the bulb (c), the inside of the rectum can be easily seen. The further introduction of the instrument is simple, because the end of the speculum can be easily pushed along the lumen of the bowel, being guided by the eye of the physician through the glass end-piece (g). The instrument should be introduced with the patient in the knee-chest position (Fig. 47). In inflammatory

and ulcerative processes in these parts of the gut, the sigmoidoscope is pushed beyond the diseased portion of the mucous membrane until normal mucous membrane is reached. Under the control of the

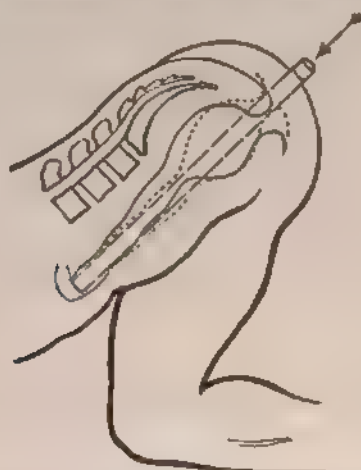


FIG. 47.—The direction of the sigmoidoscope through the sigmoid flexure, the patient in the knee-chest position. (Strauss.)



FIG. 48.—Powder blower. (Rosenberg.)

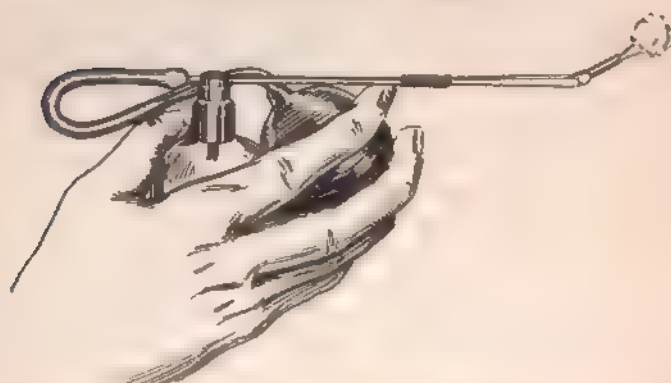


FIG. 49.—Powder blower.

eye, and while slowly removing the instrument, the entire diseased portion of the gut is covered, by means of a powder blower (Figs. 48 and 49), with a thick and even layer of some active astringent or antiseptic powder. The rectum, of course, will have had to be cleansed previously by washing it out with either water or chamomile tea. Rosenberg recommends as a medicinal application a mixture of tannic acid and subgallate of bismuth in equal parts, or a mixture of tannic acid and magnesium oxid (15 to 30 parts of the former to 100 of the latter). Zweig recommends for this method of treatment equal parts of tannic acid and bismuth subgallate, and a little common salt. The salt is intended to assist in placing the powder as high up as possible in consequence of its well-known action in stimulating reverse peristaltic movements. Bismuth subnitrate and iodoform are likewise very useful powders.

Proctoclysis.—Proctoclysis or "Murphy drip" consists of the gradual introduction of large quantities of liquid into the rectum by the drop method. The mucous membrane of the rectum absorbs water with great rapidity. The liquid must be introduced without producing overdistention, because this superinduces spasm and the expulsion of the introduced liquid. There are many instruments employed for proctoclysis. The simplest is the one devised by Murphy.¹ It consists of a fountain syringe or can with a long rubber tube attached, terminating in a vaginal hard-rubber or glass tip with numerous openings in its bulbed end (Fig. 50). The tip should be inserted into the rectum so that the bend fits closely to the sphincter.

The tube may be held in position by the use of adhesive strips bound to the thighs. The bag is suspended ten inches above the patient's thighs. When the apparatus is in place it need not be disturbed for several days. Murphy recommends for proctoclysis a solution of a teaspoonful each of sodium chlorid and calcium chlorid in a pint of water, kept at a temperature of 100° F. by applied heat in the form of hot-water bags, an enclosing can of hot water, or thermolytes. A pint and a half of water every two hours can be easily introduced in this manner.



FIG. 50.—Proctoclysis apparatus consisting of fountain syringe, large rubber tube and vaginal hard rubber or glass tip.

¹ Journal of the American Medical Association, April 17, 1909.

Ellbrecht¹ has devised an apparatus which works admirably in adults. For the maintenance of uniform heat the apparatus fulfills all indications. There is usually a great loss of heat in the rubber tubing from the reservoir to the rectum; the liquid becomes cold by the time it enters the rectum, and this retards absorption. The Ellbrecht apparatus (Fig. 51) keeps the liquid warm and insures the greatest possible absorption and least discomfort.



FIG. 51.—Metal heating chamber, block-tin lined, with opening for electric heating unit and rubber tube connection for intake and outlet of saline solution. (One-third size.)

The rectal tips shown in Fig. 52 are made in four sizes, are self-retaining, and admit a rectal tube, as shown in Fig. 53; thus the saline solution can be discharged several inches into the rectum. They prevent leakage in proctocolysis. The larger sizes are for patients with relaxed sphincter, as in advanced peritonitis, shock, severe toxemias, etc.

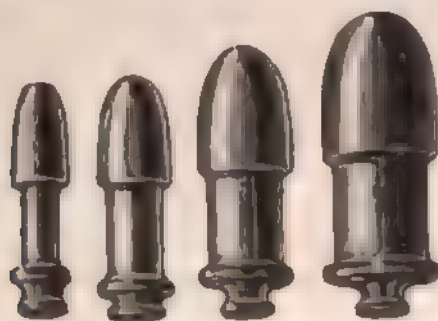


FIG. 52.—Self-retaining rectal tips, made in four sizes of hard rubber, with opening through center to admit a soft rubber rectal catheter, American size, No. 15. (One-third size.)

Fig. 54 shows a screw clamp which is placed on the rubber tubing behind the glass tube connection to catheter, to regulate the flow of water. It may be applied to any part of the tubing without disturbing the apparatus while in use, and is left wide open when the gravity method is used.

¹ Journal of the American Medical Association, November 16, 1909, p. 1249.

The electric heating arrangement is the cleanest and the most satisfactory (Figs. 55 and 56); it requires little or no attention when once started, and can be placed in the bed with the patient.



FIG. 53 Self-retaining rectal tip on catheter, showing how adjustment can be accomplished by merely drawing catheter through to desired length. (One-half size of largest rectal tip.)

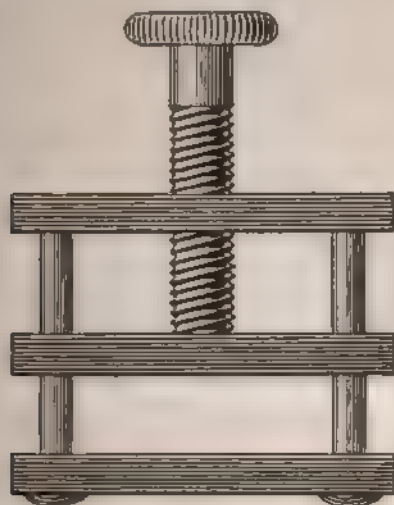


FIG. 54 Metal compressor and screw to regulate the flow of the Murphy drip.

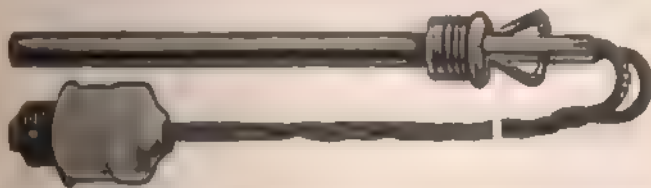


FIG. 55 Electric heating unit with socket connection and ten feet of cord. This unit may be used with either alternating or direct current, 105 to 125 volts (see Fig. 56). (One-third size.)

The same results can be obtained, however, where electricity is not available, as in rural districts or in homes where only gas is at hand, by using a Bunsen burner or an alcohol lamp in connection

with the heating chamber, which is then placed on a small table alongside the bed (Figs. 57 and 58).

A simple and inexpensive glass apparatus (Fig. 59) has been devised to be attached to an ordinary fountain syringe for regulating the flow of water by allowing it to drip. The tubing of the fountain syringe bag is attached at *a* and a short strip with rectal tips at exit *d*. The number of drops per minute can be regulated by the screw (*e*) on the compressor.

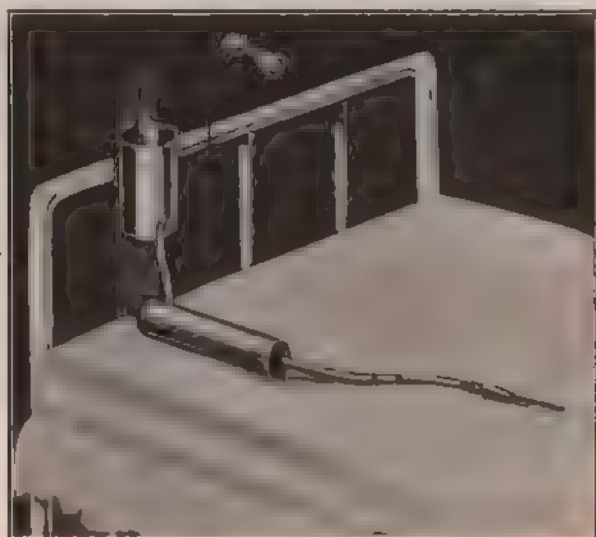


FIG. 56. Electric heater in operation, showing it properly connected. A short glass tube connects catheter to rubber tubing. (Elbrocht.)

Young¹ suggests an apparatus for continuous proctoclysis that is simple, practical and inexpensive. The chief difficulty in the management of continuous proctoclysis lies in the maintenance of the temperature of the solution at a uniform degree. The possessor of a "thermos" flask is provided with a simple means of



FIG. 57.—Heat unit for alcohol or Bunsen-burner flame with regulating piston. For use where electric current is not available (see Fig. 58). (One-third size.)

overcoming this difficulty (Fig. 60). All the additional apparatus necessary is: a U-shaped piece of glass tubing with one arm long enough to reach the bottom of the flask; three or four feet of rubber

¹ The Lancet, November 19, 1910, p. 1517.

tubing attached to one end of the glass tube and connected with the catheter by the other; and, lastly, some means of limiting the flow, such as a metal compressor on the rubber tube. The flask is filled with the solution at a temperature a few degrees above that at which it is desired to administer the injection, and suspended two or three feet above the patient's bed. The fluid is then run off by siphonage and the flow regulated by the metal compressor. The temperature of the solution will remain at a practically uniform level while the bottle slowly empties. At the rate usually advised—namely, one drop per second—only 225 Cc. (7½ ounces) are used per hour, so that a pint thermos flask will contain sufficient solution to last two hours.

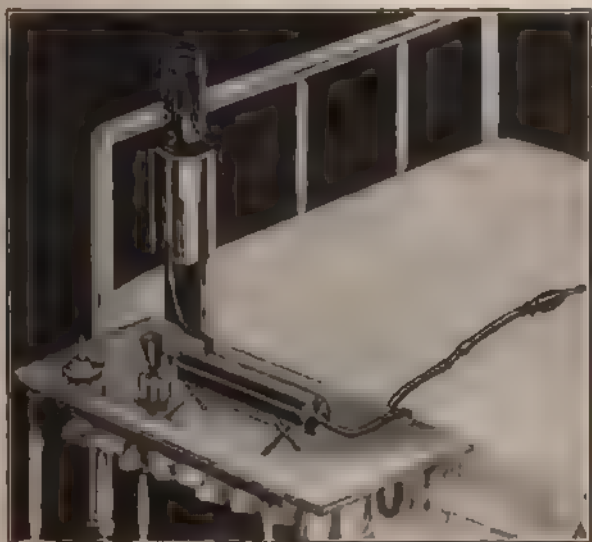


FIG. 58. Alcohol or gas heater in operation, showing it properly connected. A short glass tube connects catheter to rubber tubing. (Ellbrecht.)

Medicaments and nutrient enemata can be introduced into the rectum at will by proctoclysis. The addition of 20 per cent. of grape-sugar not only adds greatly to the nutrition but also assists in the absorption of other nutrient substances.

Nutrient Enemata.—It is now known that nutrient enemata are not completely absorbed by the rectum or colon, and the degree of absorption and assimilation declines as the period of rectal feeding is extended. When it is necessary to administer nutrient enemata, the colon should be thoroughly cleansed every day by an injection consisting of a liter (quart) of water and a teaspoonful of salt, administered early in the morning. Rectal alimentation may be given an hour later. The nutrient enema is best injected by

means of a fountain or Davidson syringe, or a plain hard-rubber piston syringe and a soft-rubber rectal tube which is introduced into the anus three to five inches. The enema (5 to 10 ounces) should be given slowly and with very little or no force, in order to prevent peristalsis, which would result in emptying the lower bowel. After the tube is withdrawn from the rectum the patient should be requested to lie quietly and to endeavor to retain the enema. Three to five such enemata may be administered daily.

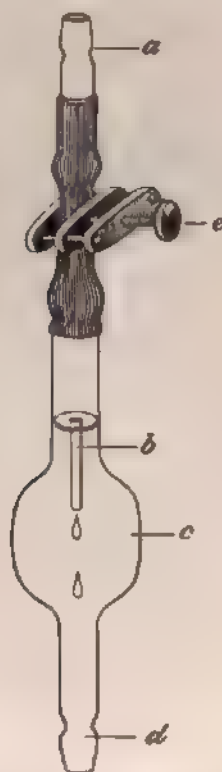


FIG. 59.—Glass attachment for proctoclysis. *a*, glass tube, *b*, glass dropper, *c*, glass nozzle, *d*, glass end for rectal tube, *e*, compressor.

When enemata are continued over a long period of time it is advisable to wash out the rectum at least once a day with warm water, soapsuds, or boric acid solution; by this means all foreign matter is got rid of, feces dislodged, and mucus and any remains of former enemata washed away.

The rectum and colon will not digest ordinary foods, but there are certain energy-producing substances that can be absorbed when given as nutrient enemata. No attempt should be made to give undigested proteins. The aminoacids should be derived from

artificially digested meat, and the vitamin should be obtained from pancreas. It should not be forgotten that alcohol is an energy food which can be utilized in rectal feeding, but it must not be given in greater concentration than 5 per cent. The composition of a nutrient enema that can be easily absorbed and duly utilized, with an energy value of 750 calories, is as follows:

	Gm. or Cc.
Glucose	50 0
Alcohol	50 0
Calcium chlorid	0 3
Sodium bicarbonate	3 0
Sodium chlorid	4 0
Ananocacids and vitamins as much as desired	
Distilled water to make	1000 0

Of this, 400 or 500 Cc. is to be given three times daily.

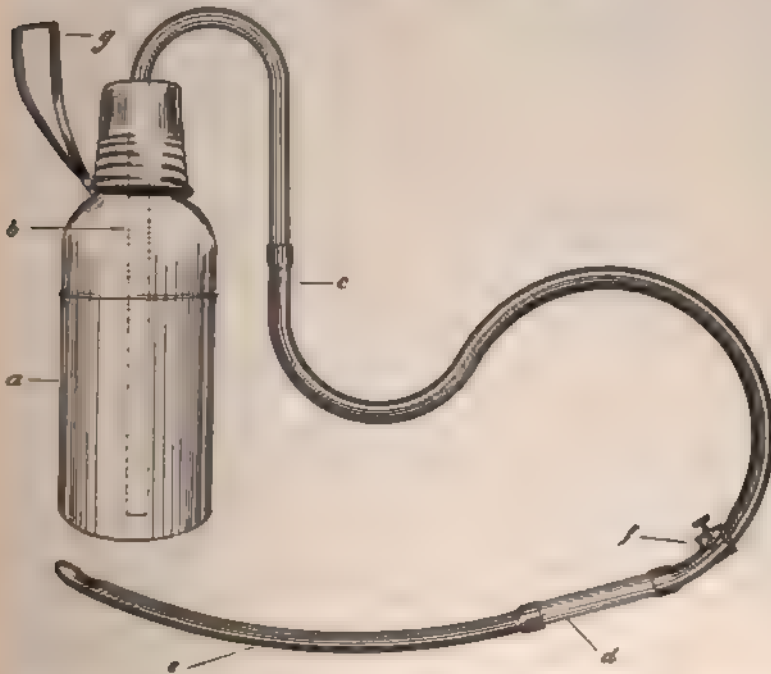


FIG. 60. —Thermos proctoclysis apparatus. a, small thermos bottle; b, U-shaped glass tube; c, rubber tubing; d, glass connection; e, catheter; f, metal compressor to regulate flow; g, leather strap to hang thermos bottle to foot of bed.

A large number and variety of nutrient enemata have been devised. Those most commonly used and shown by experience to be most satisfactory are:

Glucose or grape-sugar. A great number of calories may be introduced by this means. To $\frac{1}{2}$ liter (1 pint) of normal saline, 50 Gm. (1½ ounces) of dextrose may be added. This makes a 10-per-

cent. solution. This quantity can be introduced every six hours by proctoclysis. In twenty-four hours the patient would receive 100 calories.

Moritz recommends 15 Gm. (℥ss) grape-sugar, the same quantity of malt extract, 100 Cc. (℥ij) milk, 6 Gm. (℥iss) common salt, a wineglass of claret, and 2 or 3 eggs.

Of peptones or propeptones in the market, about two or three ounces dissolved in six to eight ounces of water may be injected. The different beef juices may also be diluted with water and injected in corresponding quantities.

Six to seven ounces of milk, one or two raw eggs well beaten in the milk, one teaspoonful of powdered sugar and one-third of a teaspoonful of common table salt, make a nutritious enema. Pancreatin may be added to facilitate assimilation (Einhorn).

Boas recommends eight ounces of milk, two yolks of eggs, common salt, one tablespoonful of claret, and one teaspoonful of flour.

The nutrient enema recommended by Ewald consists of 4 Gm. (℥x) of wheat flour stirred up in 150 Cc. (℥v) of tepid water or milk. To this mixture are added one or two eggs, 3 Gm. (℥ grains) of sodium chlorid, and 50 to 100 Cc. (℥iss-ij) of a 15-20-per-cent. solution of grape-sugar; the whole is thoroughly beaten up. Claret may be added as required.

For hospital practice Strauss recommends, chiefly on the ground of economy: 8 ounces of bouillon, 1 ounce of alcohol, 1 ounce of grape-sugar, 2 yolks of eggs, $\frac{1}{2}$ teaspoonful of sodium chlorid, 2 or 3 tablespoonfuls of gum-arabic mucilage.

Leube employs enemata consisting of well-chopped meat (5 ounces), fresh pancreas (2 ounces), and 1 ounce of fat (butter), all thoroughly mixed with about 6 ounces of water.

Should the patient experience difficulty in retaining an enema administered as advised above, 10 drops of simple tincture of opium may be added to each enema. The opium has a quiet effect upon the lower bowel, allaying any local irritability that may exist. The addition of opium to the enema has also been recommended for its influence upon the nerve control of thirst.

CHAPTER XII.

HYDROTHERAPEUTICS—MINERAL WATERS.

HYDRATIC AND THERMIC TREATMENT.

HYDROTHERAPEUTICS constitutes an important part of the treatment of diseases of the digestive organs. Water is essential to the performance of all the physiologic functions. In fact, it ranks first among the therapeutic resources. It may be used as a drink, as a spray, for lavage, or as a douche, and externally in baths, wet packs, moist rubbing, and slapping.

In the use of water as a therapeutic agent the physician should have clearly in mind the results to be attained. Cold water applied externally should have a stimulating effect, as shown by the skin reactions. In pathologic conditions of the stomach and intestine, the stimulus should be moderate in character. Weak stimuli tend to increase vitality, while stronger ones have an inhibitory effect. The physician should exercise great precaution in the treatment of digestive cases complicated with anemia, nervousness, and debility. In moist rubbing and slapping, the water should be so low as to raise body temperature, care being exercised to avoid undue heat to sensitive patients. The temperature of the water should vary from 85° to 60° F. The wet rub is best given early in the morning, inasmuch as the skin reacts best at this time, owing to the fact that it is uniformly heated on rising. The patient should not be barefooted on some non-conducting substance, such as a piece of carpet or a cork mat. A large linen sheet wrung out of water at the proper temperature is placed about him by the physician or attendant, who then proceeds to rub vigorously his back, arms, and legs. The patient, meanwhile, assists by rubbing the chest and abdomen. In a few moments this should be followed by an agreeable feeling of warmth. The patient should be dried thoroughly, and should either rest for half an hour or take a short walk before breakfast.

The "rub-off" is made as follows: The patient elevates his feet and is wrapped quickly into a moist linen sheet—one corner of which is clamped by one lowered arm while the sheet is wound around to overlap it and is clamped under the other arm also. The towel is then wrapped around the patient's trunk so that the shoulders are covered. Energetic rubbing and beating are performed by the attendant with the palm of the hand. As soon as the patient experiences a feeling of warmth he is released from the sheet and rubbed dry. In the absence of an attendant the patient may dry himself by rubbing vigorously with a Turkish towel.

Should the skin reaction following the use of the wet pack not be well marked, before another treatment the temperature of the patient should be raised either by moderate exercise or by a dry rub. If, after this procedure, the cold rub fails to bring about a skin reaction, it should be omitted; better sponge the patient off with water at a temperature agreeable to his sensitiveness—three parts water to one of vinegar, or one part alcohol to two of water, may be used. The bath may be given by applying the mixture to the whole body before attempting to dry, or a portion of the body, an arm or a leg, may be bathed and then dried, and so on until the whole body has participated in the operation.

Half Baths.—What are known as half baths have a favorable and stimulating effect upon the nervous system. The patient sits in a bath tub in which the water at 90° F. reaches as high as his umbilicus. In treating more robust patients the temperature of the water may be as low as 82° or 77° F. The patient should immerse himself to the neck in the water and return to the sitting posture. The attendant, assisted by the patient, proceeds to rub the latter vigorously. The whole bathing process should last about three or four minutes, during which time the patient should be active. On stepping out of the tub he is covered with a dry sheet and rubbed dry while either sitting on a chair or lying in bed. The bath may be followed by moderate exercise or by rest in the recumbent posture in bed. The patient should experience a feeling of comfort after each treatment.

The half bath, so called, may be varied in several ways. The patient may sit for five to ten minutes immersed to the neck in water at a temperature of 84° to 90° F. Then the water is allowed to flow out of the tub until it is at the level of the patient's umbilicus, when the attendant begins to rub him and sprinkle him with water. The half bath may be made even more intense and stimulating by allowing cold water to flow into the tub during the manipulations of the attendant. In all these procedures the head should not be allowed to become wet.

Cold Entire Pack.—What is known as the cold entire pack produces a stimulating and refreshing effect when it is employed under proper conditions. A large flannel blanket is spread upon the bed or couch, and over it a sheet which has been dipped into water of about 50° to 60° F., and which remains fairly saturated with it. After the morning evacuation of the bowels and bladder the patient is packed into the sheet and blanket so that his shoulders and arms are included in the folds. A stimulating effect is produced by removing the pack as soon as the reaction sets in. To prolong the duration of the pack beyond this point produces a quieting effect upon the patient, so that sometimes he becomes drowsy and has a desire to sleep.

Should the desired reaction not take place after the cold or wet rub, this operation may be preceded by the cold pack until the body becomes sufficiently warmed.

In this mode of hydrotherapeutic treatment of patients with gastro-intestinal disease we note at first a slowing of the temporal pulse, which soon returns to the normal rate.

To avoid hyperemia in the head or in the region of the heart, cold compresses may be applied to the head, or the cooling apparatus of Leiter (Fig. 61) may be applied over the region of the heart.

Warm Entire Pack.—By employing lukewarm water in our hydrotherapeutic treatment we may obtain a sedative effect. The blood-pressure diminishes, and with the dilatation of the bloodvessels the painful symptoms are alleviated. The patient becomes quiet, and sleep ensues.

In the prolonged warm entire pack the patient is packed in cloths which have been dipped into water of from 95° to 100° F. Since the cloths cool off rapidly, this pack must be administered quickly.

Prolonged Baths.—The prolonged lukewarm full bath acts as an agreeable sedative and hypnotic. The temperature of the water should be in the neighborhood of 95° F. The patient should be placed in a comfortable position, preferably reclining. The water should reach over the shoulders. The duration of the bath should be from five to twenty-five minutes. Should the bath be more protracted, care must be taken that the water does not cool off too much. Any kind of exertion is to be avoided, both before and after the bath. When the end desired is the induction of sleep, the tepid bath is best employed toward evening or immediately before retiring.

The prolonged baths may be medicated by the addition of various chemical agents. Sodium chlorid may be used with the water to make a 1-per-cent. or 2-per-cent. solution. Carbon dioxid at times exerts a beneficial influence upon nervous patients. The carbon dioxid bath may be prepared in private homes by the combination of sodium bicarbonate with mineral acids or with acetic acid. Oxygen baths are beneficial in the treatment of nervous dyspepsia. Such baths are prepared by adding sodium perborate and a manganese salt to the water, the sodium perborate being broken up by the manganese salt in the presence of water, with the liberation of oxygen. The immersion of the body in such an effervescing solution gives a powerful impetus to the nervous system. Many patients find the addition of 250 to 500 grams ($\frac{1}{2}$ to 1 pound) of pine-needle extract to the bath very agreeable, but the good effect is probably largely mental.

Indications.—The indications for hydrotherapeutic treatment in diseases of the digestive organs are not always clear. In a

general way hot applications tend to the diminution of pain and have an antispasmodic effect; cold applications, on the other hand stimulate.

Compresses.—Hot compresses in the form of poultices are well known to the laity. To prepare a *mashed-potato poultice*, which is one of the best forms of cataplasm, freshly cooked potatoes are placed upon a piece of cheesecloth, a portion of which is folded in the form of a sac. This bag may be closed by means of safety pins or a few stitches. The potatoes are crushed with a wooden roller, after which the poultice is ready for use. This poultice is not only the cleanest, but retains its warmth longer than any other.

Linseed poultices are made by boiling the linseed meal to a thick consistency. The mass is then folded into the cloth and used in the same manner as the potato poultice. The linseed poultice is not so satisfactory, owing to the fact that it adheres to the parts and is apt to undergo acid fermentation.

It is hardly necessary to say that cataplasms should always be applied hot and of sufficient size to cover the portion of the abdomen involved. To secure the desired effect, two poultices should be prepared, so that one may be in the steam bath while the other is doing duty on the patient. To maintain the heat double boilers are very convenient. The cataplasm is placed in a tray with a perforated bottom held above the water level in the boiler. The water may be heated by a spirit lamp or other means so that the poultice when not in use is subjected to the action of steam. The apparatus should be kept covered.

Heat may be applied to the abdominal region by means of hot towels, or heated plates well wrapped in cloth. The flat stomach-bottle, of aluminum, rubber, or zinc, is of practical value. Flat rubber bottles or boxes filled with some chemical substance are obtainable, which, after being subjected to the action of boiling water for fifteen minutes, will retain their heat for several hours. Electric warming pads and electrothermic bottles are of more recent invention. Leiter's tubes (Fig. 61) are made of tin, aluminum, or hard rubber; they are placed upon the abdomen of the patient, and hot water is allowed to run through the coil. The electrothermic bottle, electric warming pad, or Leiter's coils may be converted into moist hot compresses by encasing them in moistened folds of cloth. The temperature of the hot cataplasm must be modified according to the requirements of the patient's comfort.

When hot cataplasms are used for a long period of time, for instance in gastric ulcer, the skin over the hypogastric region should be thoroughly cleansed with soap and water and weak bichlorid solution, and a piece of flannel or linen laid over the parts and made secure by adhesive plaster. This forms a basis for the hot compress. In this way blisters from heat may be avoided.

The *Priessnitz bandage* is applied moist, and either hot or cold,

that it produces a hyperemic condition of the skin. The application of this bandage is accompanied by an agreeable feeling of warmth. The effect is sedative, analgesic, and frequently hypnotic. The Pressnitz bandage consists of a towel folded several times, dipped in warm water, and wrung out. This is placed over the stomach and covered by oiled silk or gutta-percha, with a flannel tender to retain it in place. This bandage, which should be sufficiently tight not to slip down, is adjusted at night and allowed to remain on the patient until morning. Alcohol (50 per cent.) has a more stimulating effect than water.



FIG. 61—Coiled tubing. (Leiter.)

In the treatment of nervous diseases of the gastro-intestinal tract, Waternitz recommends the use of coiled tubing, such as the Leiter cooling apparatus, in which water at a temperature of 55° to 130° F. is allowed to circulate. The coils are interposed between moist linen and woollen bandages (Fig. 61).

Douches.—Douches are often applied externally with good effect. We have the fan douche and the so-called Scotch or interrupted douche. A somewhat cumbersome apparatus is required for the application of the latter. With this apparatus the temperature may be quickly alternated from 100° to 50° F. and an interrupted jet of water thrown over the region of the stomach. We obtain

by the use of this apparatus not only alternate contraction and dilatation of the capillaries of the skin, but reflex contractions of the abdominal muscles as well. A stimulus is likewise given to the peristaltic movements of the intestine.

MINERAL WATERS.

An extensive therapy for diseases of the stomach and intestine is provided by the so-called mineral-water cures, bath cures, climate cures, and sea baths. Mineral waters are solutions of salts and gases in water. As a rule the solid constituents (salts) are present in very small amounts. In spite of the fact that these waters are among our oldest therapeutic agents, we have much to learn in regard to their physiologic action. We have as yet no well-defined scientific basis of procedure in regard to their use. Such investigators as von Noorden, Doppler, Lareche, Jaworski, Boas, and Wolf have sought to point out the direct local and systemic effects, but their results have been contradictory. In the absence of scientific data we must continue to base our use of mineral waters on empirical knowledge, controlled only by what we know of their individual constituents.

The gaseous constituents are, chiefly, carbon dioxide and sulphuretted hydrogen. Some of the waters are radio-active. The solid constituents are salts of sodium, potassium, magnesium, aluminum, calcium, iron, iodine, bromine, chlorine, and sulphur. Some of these waters have a purgative effect, some laxative, and some diuretic.

- Classification.**
1. Alkaline chlorine waters.
 2. Sodium chloride waters.
 3. Alkaline carbonated waters.
 4. Ferruginous or chalybeate waters.
 5. Bitter waters.

Alkaline Chlorine Waters.—Waters from the alkaline chlorine springs contain principally sodium chloride, sodium sulphate, sodium bicarbonate, and carbon dioxide. In the United States we have Arondack, at Saratoga, N. Y.; Bedford, at Bedford, Pa.; Berry Hill, Elkwood, Va.; Crab Orchard, Kentucky; French Lick, Indiana; Gate Springs, Tennessee; West Baden, Indiana; Hot Sulphur Springs, Colorado; Gibson's Mineral Wells, Texas; and Ferris Hot Springs, Montana. To this class belong the springs of Carlsbad, Bertrich, Marienbad, Rohitsch, Tarasp, and Franzensbad, in Europe.

Carlsbad and Bertrich are warm springs. Carlsbad is especially famous in connection with the treatment of diseases of the stomach, liver, and intestine. It has been found that a single dose or a few small doses of Carlsbad water or salt will excite a copious secretion of acid, but that larger doses continued for a longer period of time

may greatly diminish the secretion of gastric juice. Carlsbad water stimulates the liver, dissolves mucus, increases the peristaltic action of the stomach and intestine, and, owing to its warmth, diminishes gastric sensitiveness.

The individual Carlsbad springs vary in temperature. The springs with moderate temperatures are preferable to those with higher degrees of heat, especially for the treatment of ulcer of the stomach. These waters are valuable in the treatment of chronic gastritis, especially when there is a copious secretion of mucus, and in hyperacidity not of nervous origin. The employment of small doses of the very hot springs in catarrhal affections of the biliary passages and of the small and large intestine is recommended.

The good results following the use of the Carlsbad waters are partly due to the excellent diet prescribed at Carlsbad resorts.

Sodium Chlorid Waters.—In the United States are the Springs at Ballston, N. Y.; Hathorn, Congress, Kissingen, Selters, and Champion, at Saratoga, New York; Colorado Springs, Colorado; Wasatch Springs, Utah; and in Canada the springs at St. Catharines, Ontario. In Europe are the springs of Kissingen, Homburg, Soden, Wiesbaden, Pyrmont, and Mergentheim.

Sodium chlorid taken after a meal has the effect of inhibiting hydrochloric acid secretion and peptic digestion without interfering in any way with the motility of the stomach. Experiments with sodium chlorid waters, especially Kissingen and Homburg, on patients with gastric and intestinal disease, have shown, on the contrary, that in cases of gastritis with subacidity the acid secretion was increased; while in hyperacidity the employment of sodium chlorid waters is frequently followed by a marked decrease in the hydrochloric acid secretion. Their effects in subacid conditions seem to be fairly constant, that is, stimulating the secretion of free hydrochloric acid; but observers are at variance regarding their effects in hyperacidity. In subacidity with profuse mucous secretion the sodium chlorid waters cause a marked diminution in the amount of mucus.

In cases of subacid gastritis, especially in their incipency, the secretion of hydrochloric acid may be restored to normal by a course of treatment with the sodium chlorid waters. To obtain the favorable effect on the gastric secretion, the waters (Saratoga, Kissingen, Wiesbaden) should be taken on an empty stomach, and the patient should refrain from partaking of food until they have passed out of the stomach.

Alkaline Carbonated Waters.—The alkaline carbonated waters contain as their chief constituents sodium bicarbonate and carbon dioxide. The principal waters of this class in the United States are: Allouez, Green Bay, Wis.; Peerless, Saratoga, N. Y.; Vichy, at Saratoga; Skaggs, Hot Springs, Cal.; Canon City, Colorado. In Europe are Bilin, Fachingen, Neuenahr, Giesshübel, Geilnau,

Preblau, Salzbrunn, and Vichy. Owing to the fact that these waters contain sodium carbonate, they are indicated particularly in the treatment of hyperacidity, hypersecretion, and eructations. After a course of treatment with the alkaline carbonated waters, particularly Vichy, an increase in the motility of the stomach has been noted. It is important that these waters be administered warm, to lessen the sensitiveness of the stomach.

The alkaline saline waters contain, in addition to carbon dioxide and bicarbonate of sodium, small quantities of sodium chlorid. In the United States are Deep Rock Springs, Oswego, N. Y.; Manitou, Manitou, Col.; and Sheboygan, Sheboygan, Wis. They increase the secretion of gastric juice, and are indicated in chronic gastritis, slight atony, and secondary catarrhs.

Ferruginous Waters. These waters contain bicarbonate of iron and sulphate of iron. The ferruginous springs of the United States, are: Mardela, Maryland; Rock Enon, Virginia; Church Alum, Virginia; Owosso, Michigan; Sparta Mineral Wells, Wisconsin; Fruitport Wells, Michigan; Wilbot, Oregon; Millboro, Virginia; Rockbridge, Virginia; Mono Lake, California; Bath and Bedford Alum, Virginia. In Europe there are the acid iron springs of Elster and Franzensbad, and the waters of Reinerz, Rippoldsau, Schwalbach, and Bartfeld. These waters are useful in the treatment of chronic gastro-intestinal catarrh occurring in anemia and chlorosis.

Bitter Waters. Bitter waters are indicated in the treatment of diseases of the stomach, liver, gall bladder, and intestinal disease when constipation is present. They inhibit the secretion of gastric juice. Their use is contra-indicated in gastric ulcer. Among the bitter waters we have Abilena, Franz Josef, Pluto, Veronica, Arondack, Saratoga, and West Baden Sprudel.

Drinking Cures. We possess but a vague knowledge concerning the mode of action of the drinking mineral water cures, which are so often undertaken, and frequently with good results, in the treatment of gastro-intestinal disease. The beneficial effect of particular mineral waters has generally been ascertained empirically. It seems impossible to clearly understand in what peculiar manner these waters affect intestinal disorders. As in the dietetic treatment (see Chapter VII), so with the mineral waters, the purpose is to produce either a purgative or a constipating action. The former is brought about by acceleration and the latter by retardation of peristalsis. Moreover, the waters, to a certain extent, produce their effects mechanically, inasmuch as they wash out the bowel and remove the mucus, bacteria, and decomposition products.

The action of purgative waters is most easily understood. It may be stated in a general way that every one of the mineral waters is capable of exerting a purgative effect when taken cold and in

large doses. The purgative effect of the alkaline acidulous and the alkaline chlorid waters is so slight that they are not, as a rule, taken with this object in view. They contain large quantities of carbon dioxide, which increases the peristaltic action of the stomach and intestine. The sodium chlorid waters act better as purgatives, and the effect is always more marked from the cold than from the warm springs. The bitter waters are the most effective purgative waters we possess. They resemble each other in their action, which is identical with that of the saline salts, sodium sulphate and magnesium sulphate. The pure salts are not adapted to prolonged use, being apt to induce intestinal catarrh and digestive disturbances. The sodium sulphate waters are well borne for a long time, have a mildly purgative action, and in small doses contribute to the cure of chronic intestinal catarrh, because the sodium sulphate is in combination with bicarbonate of sodium, sodium chlorid, and carbon dioxide. The thermal springs of Carlsbad are particularly famous because of their good effects in chronic intestinal catarrh. The thermal springs of Bertrich and the artificial Carlsbad salt act similarly. These waters and salts, taken warm, are specially indicated in chronic gastric catarrh with accompanying constipation. They are likewise indicated in chronic catarrh associated with diarrhea; in such cases, however, they must be taken in small doses and as hot as possible. When the waters and salts of Carlsbad and Bertrich are taken cold, the purgative effect is more pronounced. Some of the waters contain more sodium sulphate than Carlsbad, and are consequently strongly purgative, especially when taken cold. These waters, therefore, are often prescribed in cases of hemorrhoids and chronic constipation in robust patients.

Two opposing theories are maintained as to the mode of action of the bitter waters. According to one theory, the liquid form of the stools is caused by transudation and dilution in obedience to the laws of osmosis. The other theory assumes that the dilution of the feces is the effect of an increased activity of the glandular elements, stimulated by improved circulation of the blood. Generally speaking, the bitter waters are not well adapted to protracted use; their continued administration is apt to cause disturbances of digestion and a diminution of the secretion of gastric juice.

Purgative waters should, as a rule, be taken in the morning on an empty stomach, one hour before breakfast. Bodily activity during the interval is an adjuvant to the efficacy of the waters. A Carlsbad cure generally requires four weeks. Such cures may be undertaken equally well at home when the domestic arrangements permit of fulfilling the requirements as to diet and rest.

Purgative mineral waters are able to produce a constipating effect when taken very hot in cases of intestinal catarrh with diarrhea or with alternating diarrhea and constipation. They have a decidedly beneficial effect on the catarrhal condition, and

thus on the diarrhea, improving the form of the fecal discharge. The other sodium sulphate springs, the sodium chlorid waters, and under certain conditions the alkaline chlorin acidulous waters, occasionally act in a similar manner. Peristalsis may also be reduced by the calcium and ferruginous waters.

The ferruginous waters, both those containing carbon dioxid and those containing sulphates, are employed in cases of chronic diarrhea and catarrh, especially when the patient is more or less anemic. The waters containing calcium, magnesium, and sodium, known as acid waters, are used in relaxed conditions of the mucous membrane, particularly in cases characterized by diarrhea. Very obstinate cases of chronic diarrhea have been cured by a sojourn at a spring rich in calcium bicarbonate. The ferruginous waters increase the amount of hemoglobin. They also increase the appetite and reduce intestinal activity. Such waters are excellent as tonics and valuable in the treatment of diarrhea.

Generally speaking, it may be stated that mineral water drinking cures are indicated in catarrhs of the stomach, biliary passages, and small and large intestine. Carlsbad waters are especially valuable in these conditions. In hemorrhoidal diseases cold sodium chlorid springs are indicated, while in chronic diarrhea hot sodium chlorid waters and the Carlsbad alkaline chlorin waters are valuable. Calcium and ferruginous waters are indicated in diarrhea. In light forms of chronic constipation the cold sodium sulphate springs may effect a permanent cure. On the other hand, drinking cures are usually ineffective in all grave and old cases of chronic constipation or chronic catarrh and diarrhea. Such cases should be placed in bed at home or in a good private sanitarium where the diet is regulated in a proper manner and where other curative agencies may be utilized. Home drinking cures may be instituted as a preliminary trial before going to the location of the springs.

All mineral waters should, by preference, be taken at the springs themselves; it is a matter of experience that the waters affect the patients more favorably when this is done. At these resorts the patient is free from excitement and business cares; his surroundings, the atmosphere and the scenery are conducive to peace of mind, and dietary regulations are more apt to be faithfully carried out than at home. The waters may, however, be taken at home if a sojourn at the springs is impossible.

As a fundamental principle no systematic mineral water treatment in gastro-intestinal disease should be recommended until a diagnosis, or at least careful examinations, including a thorough chemical analysis of the stomach contents, and in many cases of the feces too, have been made.

Mineral Baths, Sea Baths, Climatic Cures.—Bath cures are generally combined with drinking cures, but they often act beneficially

without them. They have a favorable influence on tissue metamorphosis, the activity of the skin and the nervous system, thus reflexly rather than directly modifying the gastro-intestinal disease. Salt and mud baths have been found efficacious in the treatment of gastro-intestinal affections. Waters containing enough sodium chlorid to raise their specific gravity are designated *salt*. Baths in such waters are of three kinds—weak (1- or 2-per-cent. salt), medium (up to 6 per cent.), and strong (above 6 per cent.). Three-per-cent. mineral salt solutions are employed for bathing. Sea baths have a favorable effect in inflammatory and exudative processes of the stomach and intestinal tract, as well as in cases of chronic peritonitis. Mud baths are very retentive of heat, conserving and prolonging the caloric effect upon the skin. They act locally apart from the general stimulating effect, for they seem to be able to absorb inflammatory exudates. Upon this fact depends their value in irritable conditions of the stomach, pylorospasm, gastric ulcer, gastric neuroses, and intestinal adhesions.

Many of the waters are radio-active and their virtue may be due to this fact. It is assumed that radio-activity stimulates metabolism and exercises an influence upon the internal secretory glands. Radium is a very unstable element, continually changing into another elementary body. Each atom of radium furnishes one atom of helium. Just as soon as helium is shot out, the atom radium changes to a gas to which the name radium emanation or niton has been given. Niton is a hundred thousand times more active than radium. The calculable value of a radio-active mineral water depends upon the degree of its radio-activity. It has already been found that these baths are definitely contra-indicated in cases of tuberculosis and nephritis.

Sea baths are indicated for patients with digestive disturbances due to neurasthenic conditions, gastric atony, or ptosis. Cold sea baths have a tonic effect, due largely to the salt they contain and to the movements of the waves. They stimulate gastric digestion. Well-nourished patients suffering from neuroses, as well as the anemic, do well at the seaside. Organic intestinal diseases (catarrh) are not suitable for either the seashore or the mountains, for the simple reason that the patients do not there obtain the proper diet. Every case must be decided on its own merits when a choice is to be made between sending a patient to the seashore or to the mountains. Generally speaking, patients of sedentary habits are better off in the mountains than at a seaside resort. It is self-evident that climatic cures may be combined with the drinking cure.

Change of climate and residence in high altitudes are most suitable for gastro-intestinal patients who are likewise suffering from mental overwork and nervousness.

CHAPTER XIII.

MEDICATION IN GASTRIC DISEASES.

Hydrochloric Acid and Pepsin.—Hydrochloric acid has always been regarded as an available therapeutic agent in the treatment of certain forms of gastritis, especially those characterized by deficiency of acid secretion. Clinicians, however, have been at variance in regard to the quantity that should be administered. Some have doubted the advisability of giving it in certain forms of subacidity, maintaining that in subacid conditions pepsin is always present and that the therapeutic requirements of the patient can best be met by a carefully selected dietary. A small minority greatly restrict the administration of hydrochloric acid while at the same time they abandon the use of pepsin altogether. They argue that artificial aids to digestion are not necessary, and that their habitual use is to a certain extent injurious. Every organ, we are told, is strengthened by activity and weakened by lack of exercise.

It is important, when considering the effect of hydrochloric acid, to take into account how the ingested food becomes mixed with the acid in the stomach. The mixing varies, according to whether the hydrochloric acid has been taken medicinally or secreted by the mucous membrane of the stomach itself. In artificial acidification the degree of admixture depends also upon the interval of time between the ingestion of the nutrient and the administration of the acid.

Hydrochloric acid may be taken immediately after the ingestion of food, or a few minutes later (10, 15, 20); by giving small doses at frequent intervals, which is the usual practice, the normal process of secretion of hydrochloric acid is imitated. Hydrochloric acid may be taken during the meal. Its admixture with the food is probably accomplished best when it is so taken, as it can thus reach every particle.

By experiment it has been found that hydrochloric acid taken internally has the power to stimulate the secretion of the ferments of the stomach. This is brought about by the action of the acid on the pylorus producing a *secretin*, which in turn being absorbed stimulates the secretion of gastric juice. It has also been found that ingested hydrochloric acid will directly stimulate the secretion of hydrochloric acid by the depraved gastric mucous membrane, and that the ingested acid makes it possible for the gastric mucous membrane to respond with an increased formation of acid on the

introduction of food. These statements refer to the pathologically changed gastric mucous membrane only (subacidity in gastritis).

Experimental research has shown that extensive proteolysis cannot be obtained by the administration of hydrochloric acid alone; pepsin must be given simultaneously. It was formerly assumed that the administration of pepsin was useless, since such a small amount of pepsin is necessary to proteolysis—for when free hydrochloric acid was absent, some pepsin or its precursor, pepsinogen, was found in the stomach, though only in minute quantity. In order to secure activity of the pepsin, or pepsinogen, by the introduced hydrochloric acid, it is necessary that these two become mixed; this important fact has often been totally ignored.

The administration of pepsin alone is of but little therapeutic value. After reaching the stomach it comes in contact with the hydrochloric acid at a few points only—on the outer border of the stomach contents—and can therefore exert its proteolytic action nowhere else. Pepsin given alone soon passes into the intestine without having assisted materially in the digestion of the food. It is absolutely useless to prescribe pepsin alone in cases in which hydrochloric acid is not furnished by the stomach.

Hydrochloric acid assists the intestinal digestion of protein to the extent that protein substances which have been treated previously with pepsin and hydrochloric acid can be digested much better with trypsin. Besides this, hydrochloric acid acts upon some precursor in the duodenum, producing an intestinal *secretin* or hormone, which, being absorbed, stimulates the secretion of pancreatic juice.

Hydrochloric acid, when taken internally, increases the secretion of pancreatic juice. This augmentation commences about half an hour after the introduction of the acid into the stomach, and continues for about an hour.

When large quantities of acid are given, the effect on the small intestine is the same whether the acid be administered before, during, or after meals. But when small quantities are given, it is best to give them before meals. Small quantities of acid, which *per se* have no direct effect whatever on the gastric digestion, may, when administered in this manner, exert an energetic influence on digestion in the small intestine.

It has been shown that hydrochloric acid taken by the mouth, like the natural product, prolongs the stay of the food in the stomach. This is due to a periodic closure of the pylorus brought about by the action of the hydrochloric acid on the mucous membrane of the duodenum, and takes place whether the hydrochloric acid is given during the meal or afterward.

It has been noted that hydrochloric acid is able also to stimulate the secretion of bile.

Ingested hydrochloric acid has a favorable influence on the appetite; it is therefore a direct stomachic. This effect is due to improvement in the general nutrition, and to stimulation of the peripheral nerve fibers which excite the sensation of hunger.

A number of preparations containing hydrochloric acid are at our disposal. Two solutions of the acid are official:

1. *Acidum hydrochloricum*—hydrochloric acid; 100 parts contain 31.9 parts hydrochloric acid and 68.1 parts water.

2. *Acidum hydrochloricum dilutum*—diluted hydrochloric acid; 100 parts contain 10 parts hydrochloric acid and 90 parts water.

Hydrochloric acid should be taken well diluted, through a glass tube; otherwise it decalcifies the tooth substance and irritates the mucous membrane of the mouth, pharynx, and esophagus. For the protection of healthy tissue as well as the maintenance of comfort to the patient, suitable methods of drug administration are demanded; therefore the author repeats a suggestion with regard to the administration of hydrochloric acid which his personal experience has shown meets the difficulties. He has employed this method since 1899.¹ In prescribing the acid it was at first suggested that it be taken in gelatin capsules (Fig. 62). It was found, however, that the acid penetrated the capsule too quickly. After repeated trials it was discovered that two capsules of differing sizes (the smaller one, containing the acid, encased in the larger one) would give sufficient thickness to obviate quick penetration—would, in fact, retain the acid for a long time (Fig. 63). This device gives the patient ample time for swallowing and reduces to a minimum whatever annoyance or risk is involved. The double capsule is easily constructed. A No. "0" capsule will fit into the body of a No. "00" capsule, forming with it a shell of double thickness, which, of course, offers a twofold resistance to the action of the acid (Fig. 64). The lower edge of the cap of the enclosing capsule is first moistened with the tip of the tongue, so that when it is placed over the body of the capsule it becomes immediately sealed. The patient is instructed to use an ordinary dropper for placing the hydrochloric acid in the double capsule just before taking. The capsule will hold 1 Cc. (15 minims).

Additions of other medicinal agents (except pepsin) to hydrochloric acid are not usual. To correct the taste, the acid can be given to adults in tea, with or without the addition of sugar. For children, syrup of orange is a good vehicle.

Acidol.—This is a betain chlorhydrate, prepared from molasses, which in watery solution splits up into non-toxic betain (trimethylamin acetic acid) and hydrochloric acid. It is considered harmless. Acidol without pepsin is as ineffective for good as hydrochloric acid without pepsin. Combined with pepsin it has been

¹ Charles D. Aaron: Simple Method of Administering Hydrochloric Acid, *Journal of the American Medical Association*, June 24, 1899.

introduced to the profession as acidol-pepsin tablets. A dry hydrochloric-acid-protein powder has been presented to the profession under the trade name oxyntin. It may be taken in the dry form, in a capsule or as a powder. Oxyntin contains 5 per cent. by weight of dilute hydrochloric acid, in combination with albumin, the acid loosely bound to the protein. Six grams (100 grains) of the oxyntin represent 5 minims of dilute hydrochloric acid. It is readily miscible with water, to which it imparts but a slightly acidulous taste. Compressed tablets containing 0.06 Gm. (1 grain)

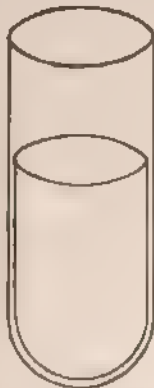
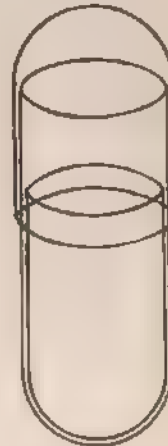
FIG. 62



FIG. 63



FIG. 64



Single "00" gelatin capsule. Inner capsule in position. Double capsule closed.

of pepsin with oxyntin are also manufactured. These preparations are not so efficacious as hydrochloric acid and pepsin in liquid form.

Fortunately the *National Formulary* gives us a number of preparations containing both hydrochloric acid and pepsin. The following is especially recommended:

	Gm. or Co.	
R—Acid. hydrochlorici	2 5	℥xl
Pepsini	21 0	℥v
Glycerini	125 0	℥iv
Aquæ q. s. ad	250 0	℥viij

Misce.

Sig —A teaspoonful to a tablespoonful in a glass of water, to be taken during meals.

Pepsin and hydrochloric acid should not be given in an alcoholic menstruum. Alcohol is a ferment poison. Various drugs influence peptic digestion. Iron is particularly detrimental. While the bitters, quinine and condurango, are tonics, they should not be given with pepsin, on account of their ferment-destroying property. It is not permissible to take pepsin in hot liquids, since a higher than body temperature destroys the activity of this ferment.

There are a number of other preparations intended to replace pepsin and hydrochloric acid. One of these is the so-called *gasterin*, or gastric juice of the dog. Pawlow has recommended this canine gastric juice as a medicinal agent. Gasterin, taken in daily doses of 250 to 500 Cc., has given good results in cases of subacidity and anacidity. The cost of the product at present is an obstacle to its general employment; besides, it is somewhat repulsive. The artificial mixture of hydrochloric acid and pepsin, fortunately, serves the same purpose.

Another preparation intended to replace hydrochloric acid and pepsin is the so-called *dyspeptine* of Hepp. This is the gastric juice of pigs. But it has been found that dyspeptine contains no hydrochloric acid whatever, that it does not digest protein, and that it is, therefore, therapeutically inactive.

Pancreatin. -Besides pepsin, another important digestive ferment is recognized by the U. S. P., namely, pancreatin. The official pancreatin possesses the property of converting twenty-five times its own weight of starch into substances soluble in water. Pancreatin should contain the pancreatic ferments: trypsin, which digests proteins; steapsin, which emulsifies fats; amyllopsin, which converts starch into sugar; and a milk-curdling ferment.

Pancreatin has marked digestive properties; in addition to its action on protein it converts all starches into sugar, emulsifies fat, and curdles milk. It is especially indicated when the stomach is deficient in secreting power. Often the gastroenterologist finds it necessary to treat the stomach as though it were a part of the duodenum. In all cases of subacidity and achylia, duodenal digestion must make up for the deficiency in gastric digestion. Patients who for years have had no severe or markedly distressing gastric symptoms may suddenly be seized with a diarrhea, due to insufficient secretion of gastric juice. When the diarrhea (gastrogenic) once develops, the irritability of the duodenum should be relieved as much as possible. The condition may be aggravated by either gastric hypermotility or pyloric insufficiency. If pancreatic digestion be instituted in the stomach, the duodenum will receive the food in a more or less digested state, and in this way irritation by fermenting foods may be largely obviated. Relieved of the irritation, the intestine, as a rule, soon regains its lost tone. Pancreatic preparations should always be given with alkalis, since the alkalis in solution in the stomach dissolve mucus. Pancreatic prepara-

tions are particularly valuable in achylia gastrica. Recent reports indicate that benefit is often derived from the administration of pancreatin in cases of alimentary anaphylaxis.

Various preparations of the pancreas have been placed before the profession under trade names. *Pankreon* is a preparation of pancreatin containing 10 per cent. of tannic acid. It is insoluble in acid media, but is split up by alkalis; it therefore passes through the stomach unchanged, exerting its digestive power in the intestine. The best preparation for us is the liquor pancreaticus of the *Natural Formulary*, which contains a small quantity of alcohol as a preservative. The formula follows:

	Gm. or Co.	gr. lxxj
Pancreatin (U. S. P.)	4 38	50j
Sodium bicarbonate	12 5	50j
Glycerin	62 5	50j
Compound spirit of cardamom (N. F.)	8 7	50j
Alcohol	8 7	50j
Purified tale (U. S. P.)	3 7	5j
Water, a sufficient quantity to make	250 0	3vuj

The dose should be a teaspoonful after each meal.

Papayotin or papain, obtained from the juice of the *Carica papaya* tree, is a digestant that is frequently used. It is said to act in both alkaline and acid media.

Pineapple juice possesses the power of assisting in the digestion of proteins. Boiling or heating, as in the process of canning pineapples, destroys the digestive power of the juice. Taken raw or in the natural state, this ferment is active in either acid or alkaline media, but not in neutral solutions.

The diastatic ferments are suggested in those cases in which there is defective secretion of these normal enzymes. The ptyalin of the saliva, however, is rarely absent. When diastase is indicated, the best form of this ferment seems to be that present in pancreatin. Vegetable diastase, as found in extract of malt, is sometimes employed. There are also available many proprietary preparations of animal and vegetable diastases. Diastase should always be prescribed with alkalis, or during the meal, before free hydrochloric acid begins to accumulate in the stomach. The giving of diastatic ferments does not remove the cause of indigestion, and therefore is not resorted to as often as formerly, although it will often give symptomatic relief.

Alkalis.—While the administration of hydrochloric acid for therapeutic purposes dates from the discovery of the fact that the acidity of the gastric juice is due to hydrochloric acid, the administration of alkalis has been practiced since an early period in the history of medicine. It has long been known that alkalis exert a beneficial influence over certain diseases of the stomach. Sodium bicarbonate is preferred to the potassium salt in disturbances of the stomach when there is much pain and a tendency to nausea

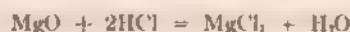
accompanied by a gouty or rheumatic diathesis. Sodium chlorid in large doses is a safe and easily available emetic. In the intestinal canal the sulphate and the phosphate of sodium act as hydragogue purgatives. They also act as stimulants to the intestinal glands, and are being constantly absorbed and excreted, reabsorbed and reëxcreted, in their course along the bowel.

Among the alkalis the Carlsbad waters or those of the Congress and Hathorn Springs of Saratoga, N. Y., and the Bedford Springs in Pennsylvania come in for consideration. The artificial Carlsbad salt constitutes an efficient substitute for the more expensive natural salt. The composition of the artificial salt is as follows (*German Pharmacopœia*):

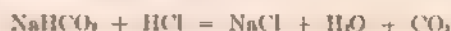
Sodium sulphate, dry	44 parts
Potassium sulphate	2 "
Sodium chlorid	18 "
Sodium bicarbonate	36 "

This salt may be administered in doses of one or two dessert-spoonfuls in half a pint of water, in hyperchlorhydria, hypersecretion, or gastric ulcer, the purpose being to neutralize the excessive secretion of hydrochloric acid. It has been used with greater or less success in gastritis and enteritis.

The alkalis are commonly divided into two groups—(1) alkaline earths; (2) alkaline carbonates. Of the *alkaline earths*, magnesium oxid or calcined magnesia is perhaps most important, as well as being the one that is generally employed when alkalis are indicated. Magnesium oxid is prepared by exposing magnesium carbonate to a dull red heat. It is a white, very light powder, sparingly soluble in water. The dose is 0.3 to 2 Gm. (5 to 30 grains), repeated if necessary. In selecting an alkali, that which liberates the least amount of carbon dioxid in the neutralization process should be chosen, inasmuch as the distention of the weak muscular walls of the stomach by gas is very annoying to the patient, not to say dangerous on account of the pressure exerted in the region of the heart. Of these alkalis, magnesium oxid or the light calcined magnesia occupies the first place. The chemical reaction that takes place when magnesium oxid is brought into contact with free hydrochloric acid in the stomach is expressed as follows:



Belonging to the *alkaline carbonates* are sodium carbonate and sodium bicarbonate. Sodium bicarbonate combines with hydrochloric acid to form sodium chlorid, water, and carbon dioxid. The chemical equation is as follows



Carbonate of sodium is used but rarely, owing to its caustic effect on the mucous membrane.

The best time for administration of alkalis in hyperacidity is from one-half to one hour after meals, at the height of digestion. The subjective symptoms of the patient, as gastralgia, eructation, pyrosis, distention, constitute very good guides as to the proper time for administering the alkali. Owing to the variable quantity of hydrochloric acid found in the stomach in the absence of food in cases of hypersecretion, alkalis should be administered before meals in such cases, in order to insure salivary digestion in the stomach. Amylolytic may be greatly assisted by the administration of a glass of Saratoga, Vichy, or sodium bicarbonate solution, 4 Cc. (1 dram) to one-half pint of water, before meals.

In treating hyperacidity or hypersecretion the magnesium salts are to be preferred to the other alkalis, especially when constipation and flatulence are pronounced.

Bismuth.—The bismuth preparations are derived from the metal itself. Among the salts used most commonly in the treatment of gastric affections are (1) bismuth subnitrate, (2) bismuth salicylate, (3) the subcarbonate of bismuth, and (4) bismuth subgallate. Bismuth subnitrate is a white, odorless powder, with a high specific gravity, insoluble in water, and very faintly acid. The usual dose is 0.3 to 2 Gm. (5 to 30 grains). It may be employed, however, in much greater quantity for the purpose of rendering the stomach or intestinal canal opaque for roentgenography, though the subcarbonate is better.

The salicylate of bismuth is prepared by the interaction of bismuth nitrate and sodium salicylate. It is obtained as a whitish and amorphous powder insoluble in water, and is administered in doses ranging from 0.3 to 2 Gm. (5 to 30 grains).

The subcarbonate of bismuth is the result of a chemical reaction between bismuth nitrate and ammonium carbonate. It also occurs as a heavy white powder, insoluble in water. The dose is 0.3 to 2 Gm. (5 to 30 grains).

Bismuth subgallate is a fine, bright yellow powder, odorless, unaffected by exposure to light. It is recommended in gastric fermentation associated with diarrhea. The dose is 0.3 to 0.6 Gm. (5 to 10 grains).

The bismuth salts are all insoluble in the stomach, where they exert a sedative and astringent action, either by their effect upon the nerve endings or the bloodvessels in the stomach walls or by coating the mucous membrane. They are used more or less extensively in the treatment of vomiting and pain due to gastric catarrh or to irritants such as alcohol, and are important therapeutic agents in the treatment of gastric ulcer and gastric carcinoma. These salts often exert a favorable influence on so-called nervous or reflex vomiting in cases of pregnancy or hysteria with true gastritis.

Bismuth salts were early known to be efficacious in gastric diseases. They were at one time abandoned on account of the frequency with which poisoning resulted, due to impurities, for the most part from arsenic. Since, however, by improved methods of manufacture an absolutely innocuous drug has been produced, the bismuth salts are again widely employed—both for their anodyne and for their antacid effects. They ameliorate or promptly relieve pains, cramps, burnings, and sensations of weight, referable to the stomach. In certain forms of gastric neurosis, such, for example, as nervous dyspepsia and gastric crises of central origin, any relief obtained by the administration of bismuth is at best only temporary. The bismuth salts, especially bismuth subnitrate, are among our best agents in the treatment of gastric ulcer; owing to the soothing and astringent influence which they exert, the lesion is in many instances healed. The subnitrate of bismuth seems to exert a very marked influence upon such reflex symptoms as retching, vomiting, and eructations. The drug has been employed with advantage in hematemesis.

It is assumed that bismuth subnitrate liberates some of its nascent nitric acid, which acts as an astringent and antiseptic on the mucous membrane of the gastro-intestinal tract. The inefficiency of bismuth subcarbonate is supposed to be due to the absence of this acid. Bismuth forms a protective layer over gastric erosions and ulcers, thus preventing existing lesions from coming in direct contact with the acid gastric juice (see page 504).

Strychnin and the Bitters.—Strychnin sulphate is prepared from *nux vomica*. It occurs in colorless, odorless, prismatic crystals, and has an intensely bitter taste. It is sparingly soluble in cold water, more soluble in boiling water. The dose is 0.001 to 0.003 Gm. ($\frac{1}{80}$ to $\frac{1}{20}$ grain). Strychnin and *nux vomica* possess the properties of stomachics. The so-called vegetable bitters or stomachics taken into the mouth stimulate the nerves of taste, producing thereby several reflex effects which are of prime importance in the promotion of digestion. The flow of saliva is increased, to the advantage of diastatic digestion, and the vessels and glands of the stomach are excited through the central nervous system.

In pyloric insufficiency large amounts of strychnin may be given, beginning with small doses and gradually increasing until 0.01 Gm. ($\frac{1}{20}$ grain) can be given three times a day. The alkaloid is useful in the treatment of gastralgia, in which condition 0.001 Gm. ($\frac{1}{80}$ grain) of the sulphate may be given hypodermically.

The class of bitters includes also such drugs as calumba, quassia, cinchona, gentian, orange, and condurango. A distinction has been drawn between simple bitters and true stomachic drugs. The former stimulate the appetite, while the latter (the complex bitters) stimulate not only the appetite, but the secretory and motor functions of the stomach as well. How the stimulating

effect upon the appetite and digestive functions is brought about is not definitely known. These remedies are indicated, as a rule, when the appetite is poor. Loss of appetite usually accompanies those gastric conditions in which the secretion of gastric juice is more or less reduced. When gastric secretion is normal the value of the bitters is questionable.

Alcohol is said to act as a stimulant to gastric secretion, but it has no effect whatever in the production of pepsin. When alcohol is introduced by the rectum as an ingredient of a rectal enema it has the power of stimulating gastric secretion. The bitter tonics have been given as tinctures, and it may be that the alcohol in the tincture stimulated the secretion of gastric juice instead of the bitters themselves. According to the investigations of Pawlow, meat juices, raw meat, meat broth, meat extractives, peptones, milk, and gelatin, as well as large quantities of water, have the effect of stimulating gastric secretion.

Condurango bark was declared at one time to possess peculiar efficacy in the treatment of gastric carcinoma. Since 1874, when Friedrich first called attention to condurango as a therapeutic agent in carcinoma of the stomach, it has been widely administered, but not with the results claimed by Friedrich. While this drug has no specific action on carcinoma, it is of some value as a stomachic. Condurango is best administered in the form of a decoction.

R—Cort condurango 15 parts
 Macerate for twelve hours with distilled water . . . 360 "
 Then evaporate down until, when strained, it equals 180 "
 Sig.—A tablespoonful twice daily.

Orexin (phenyldihydrochinazolin hydrochlorid) was introduced to the profession by Penzoldt, who claimed that it possessed the property of inducing hunger and improving the appetite. Orexin has been found to be an irritant to the gastric mucous membrane. For the original product a basic orexin was later substituted, and still later the tannate; some of the disagreeable features of the preparations have been eliminated by administering it in capsules. The dose should be followed by a large draught of water. The best results are obtained from orexin when it is administered in a dose of 0.3 Gm. (5 grains) once a day, preferably at ten o'clock in the morning, and continued for about five days. The special indications for its administration are gastric atony and acute gastritis. It is contra-indicated in such conditions as gastric ulcer, hyperacidity, hypersecretion, and other irritable conditions of the stomach.

Silver Nitrate.—Silver nitrate is prepared by the interaction of silver and nitric acid; it occurs as colorless tubular rhombic prisms. It is soluble in half its weight of water. Owing to the readiness with which this salt combines with chlorids, all solutions should

be made with distilled water, and when they are to be preserved for any length of time they should be kept in amber-colored containers. Silver nitrate is slightly soluble in 90-per-cent. alcohol. The incompatibles of this salt are alkalis and the carbonates, chlorids, acids (except nitric and acetic), potassium iodid, solutions of arsenic, and astringent infusions. In the stomach nitrate of silver is decomposed by hydrochloric acid and mucus, and cannot act as an irritant upon the mucous membrane unless administered in toxic doses. Baihakoff found that silver nitrate has the property of increasing the acidity of the gastric juice, especially in cases in which there was hyperacidity before the use of the drug. According to this, silver nitrate is contra-indicated in hyperacidity, hypersecretion, and peptic ulcer. The silver salts are indicated rather in the treatment of the subacid conditions which usually accompany chronic gastric catarrh. In chronic gastritis the power to digest proteins is somewhat diminished, so the effect of the silver salts by way of increasing gastric secretion meets the therapeutic requirements in this class of cases. Silver nitrate has been found to have an antiecatarrhal action on the gastric mucosa in gastritis. The drug exerts an antifermentative influence also, inhibiting the development of gases, belching, and eructations. Experiments have shown that silver nitrate possesses the power of increasing gastric motility. The test breakfast has been found to leave the stomach within a shorter interval when nitrate of silver is administered than when no medication is employed.

The dosage of nitrate of silver should be so regulated as to meet the requirements of the individual case or particular stage in the progress of the disease. Large doses, 0.03 Gm. ($\frac{1}{3}$ grain), administered three times a day, increase the flow of gastric juice; usually, however, this effect may be accomplished with doses as small as 0.002 Gm. ($\frac{1}{50}$ grain) given three times a day. The physician administering nitrate of silver should be on his guard against argyria.

Gastric Sedatives.—Among gastric sedatives are drugs which reduce the excitability of the vomiting center. In this class are amyl nitrite, nitroglycerin, opium, chloral hydrate, the bromids, and dilute hydrocyanic acid. As sedatives to the afferent nerves of the stomach may be mentioned hot water, ice, dilute hydrochloric acid, carbon dioxid, bismuth, dilute alkalis, opium, ipecac, and calomel in small doses.

Amyl Nitrite.—Amyl nitrite occurs in the liquid form, being chiefly an isoamyl nitrite. It is an ethereal liquid of a yellowish color, fragrant odor, and faintly acid reaction, readily soluble in 90-per-cent. alcohol, but almost insoluble in water. It is administered as a vaso-dilator in circulatory disturbances, in the form of vapor (inhalation) from an amyl nitrite pearl, or thin glass shell, which is crushed by the patient in a handkerchief. The

dose internally as a gastric sedative is one-half to one minim in rectified spirit.

Nitroglycerin.—Nitroglycerin, trinitrin, or glonoin, is a colorless oily liquid with a sweetish taste, very slightly soluble in water, but freely soluble in fats, oil, alcohol, or ether. Its uses are similar to those of amyl nitrite. The dose is one-half minim to two minims.

Chloral Hydrate.—Chloral hydrate is prepared from chloral by the addition of water. The drug occurs in colorless crystals, soluble in an equal quantity of distilled water, 90-per-cent. alcohol, or ether. It is likewise soluble in four parts of chloroform. The dose is 0.3 to 1.2 Gm. (5 to 20 grains) in solution. While the chief use of chloral hydrate is as a hypnotic, it has been found valuable for allaying vomiting or irritability of the stomach, owing to its sedative effect on the vomiting center.

Bromids.—The bromids are gastric sedatives, inasmuch as they act as depressants not only to the brain and spinal cord but to the peripheral nerves.

Dilute Hydrocyanic Acid.—Dilute hydrocyanic acid is an aqueous solution, a colorless liquid, faintly acid in reaction, with a specific gravity of 0.997. It is incompatible with the salts of iron, copper, and silver. Its chief use is as a sedative to the nerves of the stomach. It is employed to relieve gastric pain and allay vomiting in ulcer and in reflex and other nervous disorders of the stomach. In all probability the greater share of the influence exerted by this drug on the conditions named is exerted by way of the medulla oblongata. Hydrocyanic acid is speedily disseminated throughout the tissues, selecting for its action the nerve structures. The drug also acts as a cardiac sedative, especially in heart conditions resulting from derangement of the gastric function. The dose of the dilute acid is 0.06 to 0.2 Cc. (1 to 3 minims).

Cannabis Indica.—*Cannabis indica* (Indian hemp) is prepared from the dry tops of *Cannabis sativa* grown in India. Among the preparations prescribed are the alcoholic extract, dose 0.015 to 0.06 Gm. ($\frac{1}{4}$ to 1 grain), and the tincture, dose 0.3 to 1 Cc. (5 to 15 minims). The drug may be used internally as a corrective of griping purgatives such as podophyllin and colocynth. Large doses produce a peculiar species of intoxication, involving disordered consciousness of personality, locality, and time. The local effect upon the stomach is that of a sedative. The drug is said to provoke a ravenous appetite at times. American cannabis, from *Cannabis sativa* grown in America, has the same effect as the Indian drug, and its preparations are administered in the same dose.

Cocain Hydrochlorid.—Cocain hydrochlorid, the salt of cocain most frequently employed for medicinal purposes, is obtained from the leaves of the *Erythroxylon coca*. The salt is in the form of fine crystals that are soluble in half their weight of cold water and in four parts of alcohol. With water, cocain₂hydrochlorid

forms a colorless solution, neutral in reaction; the solution has a bitter taste, causing tingling of the tongue, soon followed by numbness. The dose is 0.01 to 0.03 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain). Cocain hydrochlorid as a local anesthetic is well known. The effect of the drug is confined to mucous membrane and the deeper tissues; the skin is peculiarly exempt. Cocain hydrochlorid may be used as a local sedative in all irritations of the stomach. In vomiting accompanied by pain it has been found extremely valuable.

Apothesine, a recent discovery is administered by mouth in the treatment of nausea and gastric pain; it acts as a local anesthetic in doses of 0.015 to 0.03 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain).

Gastric Anodynes.—*Chloroform*. Five or six drops of chloroform on sugar or ice is useful in the treatment of selected cases of gastralgia. Chloroform has been found not only to afford temporary relief from pain, but to arrest the course of the general disease. Chloroform water (1 : 150) can be administered every hour in tablespoonful doses. Its action is that of a local sedative and antiseptic. Small doses of chloroform have been found capable of arresting vomiting in gastric ulcer. Chloroform may be administered conveniently with bismuth.

Orthoform-nov.—Orthoform is a methylaminoparaoxybenzoate. It is a fine, whitish, odorless, tasteless powder, sparingly soluble in water, and is credited with possessing local anesthetic and antiseptic properties. It is said to be non-toxic. Its analgesic action is manifest only when the drug comes into direct contact with the exposed ends of nerves. Orthoform as a local anesthetic resembles cocain somewhat, but differs from the latter in the fact that, owing to its insolubility, it does not penetrate the tissues. It has been prescribed extensively, to be taken by the mouth, for the relief of the pain of gastric ulcer; and the fact that it does not get below the surface, and therefore cannot relieve any but superficial pain, makes it useful as a diagnostic agent. When relief of gastric pain follows its internal administration, this fact is considered an indication of the presence of ulcer of the stomach. The internal dose is 0.5 to 1 Gm. (7 to 15 grains) in the form of a mixture.

Anesthesin. Anesthesin is ethyl-paraminobenzoate, or the ethyl ester of paraminobenzoic acid. It occurs as a white, crystalline, odorless and tasteless powder, which produces a sensation of numbness when placed on the tongue. It is with difficulty soluble in hot water, and almost insoluble in cold. In six parts of alcohol or ether it should form a clear, colorless, neutral solution. It may be sterilized in oil solutions without undergoing decomposition. Anesthesin was introduced to the profession as a local anesthetic resembling orthoform in its action. It does not penetrate mucous membranes, and, being insoluble in water, cannot be administered hypodermically. It has been prescribed for the relief of pain in gastric ulcer and gastric carcinoma and in various forms of gastralgia. The dose is 0.3 to 0.5 Gm. (5 to 7 grains) in capsule.

Drugs Used Incidentally in Gastric Disorders.—*Atropin, Pilocarpin, and Nicotin.*—We have a number of drugs which are used largely for their indirect effect in the treatment of gastric conditions. Atropin, the alkaloid of belladonna leaves or root, performs an important rôle when there is an excess of secretion. Belladonna produces a slightly anodyne effect when taken into the stomach, and has been used to relieve some forms of gastralgia. It has been found especially valuable in cases of vagotonia. The hypodermic use of atropin in hyperacid conditions was first recommended by Riegel. Owing to the fact that in order to obtain inhibition of gastric secretion the dose of the drug must be somewhat large, there is more or less danger of poisoning from the use of it. Regarding the action of atropin, pilocarpin, and nicotin, it may be said that atropin in small doses injected directly into the blood or into the salivary gland duct prevents the action of the chorda tympani, thus producing inhibition of the salivary secretion; it apparently paralyzes the endings of the cerebral fibers in the glands. Pilocarpin is mentioned, owing to the fact that its effect upon the secretory mechanism is exactly opposite to that of atropin. From the minutest doses of pilocarpin we get a continuous secretion of saliva; it is supposed that the drug stimulates the endings of the secretory fibers of the salivary glands. Pilocarpin and atropin are to a certain extent physiologic antagonists. Nicotin in its effect upon salivary secretion differs from both of the other two; it inhibits the action of the secretory nerves by paralyzing the connections between the nerve fibers and the ganglion cells. These drugs are valuable in the treatment of the neuroses associated with "vagotonia" and "sympathicotonia" (see page 388).

Eumydrin.—Eumydrin (atropin methyl nitrate) is the nitrate of methylated atropin. It is similar in its action to atropin, but reputedly much less toxic, and may therefore be given in larger doses. According to Schoenheim, eumydrin is fifty times less poisonous than atropin sulphate, and, owing to the introduction of the methyl group, is entirely devoid of any action upon the central nervous system. It is therefore able to act more powerfully upon the peripheral nerve endings and secretory glands. The dose of eumydrin is 0.001 to 0.003 Gm. ($\frac{1}{80}$ to $\frac{1}{20}$ grain).

Epinephrin. Epinephrin (adrenalin) is a substance obtained from the suprarenal glands of sheep or other animals. It is an alkaloidal product, slightly alkaline in reaction. It is a powerful styptic, exercising a constricting effect on the bloodvessels, with a consequent raising of blood-pressure. Hypodermically the dose is 0.06 to 1 Cc. (1 to 15 minims) of the 1:1000 solution diluted with sterile water. Epinephrin has been employed to arrest gastric hemorrhage, being administered by mouth in doses of 20 to 30 drops of the 1:1000 solution three or four times a day. No untoward results seem to follow the prolonged administration of the drug in these cases.

Antiseptics.—Among the drugs used as antiseptics for the stomach we have resorcinol, phenol, and the salicylates.

Resorcinol.—Resorcinol is a phenol derivative which occurs in white, lustrous crystals with a sweetish, pungent taste. It is soluble in equal parts of water, twenty parts of olive oil, or half its weight of alcohol. Resorcinol is essentially an antiseptic, disinfectant, analgesic, and hemostatic, being non-irritating in solutions of 2 to 10 per cent. The dose is 0.12 to 0.25 Gm. (2 to 4 grains) after meals, in pill or capsule.

Phenol.—Phenol, or carbolic acid, is obtained by the fractional distillation of coal tar. It occurs in colorless hygroscopic crystals, soluble in 12 parts water, freely soluble in glycerin. Phenol is an excellent antizymotic. The manner in which it performs the function of antizymosis is not well understood. In vomiting due to a neurosis or gastric irritation 0.03 to 0.12 C.c. ($\frac{1}{4}$ to 2 minims), well diluted, depresses the sensory nerves of the stomach.

Salicylates.—As antizymotics, the salicylates, particularly sodium salicylate, retard the fermentation of milk in the stomach and promote its digestion. Given to patients with gastro-intestinal disease, they destroy the fetid odor of the breath as well as that of the feces. The drug should, as a rule, follow the administration of a purgative in order that the colon may be kept free, inasmuch as gastric disturbances are often caused by fecal impaction. Acetyl-salicylic acid will allay irritation, mitigate pain, and reduce congestion of the gastric mucosa. It must be given in small doses, 0.5 Gm. ($7\frac{1}{2}$ grains) with meals. The salicylates are valuable for controlling certain reflex symptoms of gastric origin, such as flushing of the face, congestive headache, vertigo, and insomnia of gastric origin. Salicylic acid is said to reduce gastric secretion by about one-half, while it increases biliary secretion to the extent of 20 to 80 per cent.

Iodin.—Tincture of iodine is occasionally employed in the treatment of gastric ulcer, both for its anodyne effect and as a stimulus to healing. It is likewise a valuable antiseptic. Administered in drop doses, freely diluted, it has proved efficacious in some cases of vomiting of pregnancy that had resisted other measures.

Hydrogen Peroxid. Hydrogen peroxid is prepared by the interaction of water, barium peroxid, and a dilute mineral acid, at a temperature below 10° C. It is a colorless, odorless liquid, with a slightly acid taste. Heat decomposes it into water and oxygen. Aqua hydrogenii dioxidi, U. S. P., should contain 3 per cent. of absolute hydrogen dioxid. It is a powerful oxidizing agent, possessing marked disinfectant properties. Rinsing the mouth with a 1-per-cent. solution of hydrogen peroxid has been found to cause marked increase in the secretion of saliva. Internally administered, hydrogen peroxid has been found to reduce the total acidity of the gastric secretion, especially the proportion of

free hydrochloric acid. When the purpose is to reduce the acidity within normal bounds, hydrogen dioxid should be given like a mineral water on the fasting stomach in the morning in the proportion of 1 to 3 Cc. in 200 to 300 Cc. of water. In hyperacidity and acid fermentation hydrogen peroxid may be used in 0.25 to 0.5 per cent. solution for washing out the stomach. The drug is useful in the treatment of hyperacidity, hyperchlorhydria, ulcer, and spasm of the pylorus.

Magnesium peroxid is placed on the market under the trade name of magnesium perhydrol. In the acid gastric juice magnesium peroxid is converted into magnesium salts and peroxid of hydrogen. Upon this latter substance its therapeutic value depends. It can be administered in tablet form, 0.5 Gm. ($7\frac{1}{2}$ grains) containing 25 per cent. of magnesium peroxid.

Emollients.—*Olive Oil*.—Cohnheim was among the first to draw attention to the value of oil in the treatment of gastric affections. He mentions a case of probable traumatic ulcer of the stomach so painful that the patient avoided food, in which complete relief of the distressing symptoms followed the administration of a wineglass of olive oil before meals. Amelioration of symptoms by the use of olive oil has been reported even in carcinoma of the stomach; satisfactory results are frequently obtained in the treatment of benign pyloric stenosis. Olive oil decreases gastric acidity and retards the evacuation of the stomach. This oil is useful in the treatment of spasm, pain, and hyperacidity, as well as for increasing the nutrition of the body. Permanent cures have been reported in cases of spastic stenosis, fissures and erosions of the pylorus, ulcer, and gastritis (see pages 481 and 484).

Olive oil is laxative and nutritious. During its use patients may pass lumps of white fat composed of undigested palmitin. In doses of one-half to three ounces it has been known to relieve obstructive jaundice. It is a valuable remedy in hepatic colic. In gallstone disease large doses (from three to five ounces) of olive oil will frequently mitigate pain, though not, as supposed by some, bring about a disintegration of the concretions.

CHAPTER XIV.

MEDICATION IN INTESTINAL DISEASES.

THE medicinal treatment of diseases of the intestine is based upon the same principles as the dietetic treatment. It is necessary in some cases to produce constipating effects and in other cases to induce purgation. Generally speaking, medication is only to be employed when dietetic measures are found to be inadequate.

INTESTINAL SEDATIVES.

The sedatives exert their characteristic effects:

1. By putting the intestine at rest through nervous channels (narcotics).

2. By their direct influence on the intestinal mucous membrane affected by hypersecretion or hyperemia (astringents and protectives).

3. By inhibiting the putrefactive or fermentative processes of the intestine, thus producing indirectly a beneficial effect on the mucous membrane (antiseptics and antifermentatives).

Narcotics.—The preparations of opium are the sovereign remedies for alleviating intestinal pains and the profuse acute diarrhea that accompanies them. Opium contains a variety of vegetable substances, with a large number of alkaloids combined with meconic acid. The most important of these alkaloids is morphin. Medicinally only such specimens of opium should be used as are rich in morphin (at least 10 per cent.) and poor in the other alkaloids (4 to 5 per cent.). The effect of small doses of opium is therefore qualitatively nearly identical with that of morphin. Opium is preferred to morphin in intestinal diseases because it is slowly absorbed, and the effects are therefore much milder and more gradual, less poisonous, and more lasting. The slow absorption of opium is due to the fact that it contains a large amount of resinoid colloidal ingredients, in which the alkaloids seem to be embedded, and which are slowly dissolved in the intestine. The drug paralyzes the peristaltic movements of the intestine. The effect of opium on peristalsis is twofold: by stimulation of the inhibitory centers through the splanchnic nerves, and by direct action on the nerves and muscular structures in the wall of the intestine itself. This desirable effect is not to be obtained by means of any other drug. If there is pain in the abdomen, opium is a powerful anodyne; the

intestine is nearly always quieted promptly, and the pains disappear. We may say, therefore, that opium is indicated in abnormally strong peristalsis accompanied by diarrhea, and to relieve enteralgias and inflammatory processes. It is most frequently used to modify acute diarrhea, particularly in cases of acute intestinal or gastro-intestinal catarrh. The intestine, however, should first be thoroughly cleaned out, so that the irritating material that caused the catarrh may be entirely eliminated. After this evacuation, either resulting spontaneously or by the aid of calomel or castor oil, is the time for the administration of opium. At this period the feces are entirely liquid and the bad odors absent. Tenesmus occurring simultaneously with the diarrhea is a further indication for opium. The opium should be given freely and energetically at first, but only for a short time. Usually one day of it is sufficient.

Opium is contra-indicated in chronic diarrhea, chronic catarrh, ulcers, gastrogenic and nervous diarrheas. In such cases it would probably keep the bowel quiet for a short time, allowing the retention of decomposing and fermenting fecal masses. Opium is furthermore contra-indicated in old age and childhood, as during these periods there often exists great hypersensitiveness toward even small doses of this drug.

Opium may be administered in the form of *extractum opii*, containing 20 per cent. of crystallized morphin, a dry, reddish-brown extract, which may be prescribed in doses of 0.03 to 0.06 Gm. ($\frac{1}{4}$ to 1 grain) as a powder or pill or in a mixture. *Tinctura opii* (laudanum) is frequently used; it is an alcoholic solution of opium which may be given in doses of 0.2 to 1.2 Cc. (3 to 20 minims). *Tincture opii camphorata* (paregoric) is a popular remedy in doses of 4 to 8 Cc. (1 to 2 drams). Sometimes Dover's powder (*pulvis ipecacuanhæ et opii*) may be given; it is prescribed in doses of 0.25 to 1.5 Gm. (4 to 25 grains). If necessary, *extractum opii* may be given in the form of suppositories containing 0.03 Gm. ($\frac{1}{4}$ grain), but their action is slower.

A preparation of opium under the trade name *pantopon* (*pantopinum hydrochloridum*) is said to contain all the alkaloids of opium in combination with hydrochloric acid, which renders it easily soluble. It was prepared on the suggestion of Sähli, with reference to the fact, verified by clinical experience, that the entire alkaloids of the poppy plant, contained in opium, have a more marked sedative, hypnotic and constipating effect than morphin alone, while their disagreeable after-effects are much less than those of morphin. *Pantopon* is given internally in doses of 0.005 to 0.02 Gm. ($\frac{1}{20}$ to $\frac{1}{5}$ grain). Subcutaneously injected, in 2-per-cent. solution, it produces no irritation whatever.

Papaverin is a white alkaloid from opium which has a marked antispasmodic effect in doses of 0.008 to 0.016 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain). The *papaverin* group produces relaxation in tonus and inhibition

of peristaltic movements of all smooth muscle organs. This action is the direct opposite of that of morphia, which tends to raise smooth muscle tonus and stimulate its contractions. Macht discovered that the inhibitory and tonus-lowering properties of papaverin were due to the benzyl grouping of the papaverin molecule. By further investigation he found that benzyl benzoate produced the papaverin effect without being itself very toxic. Benzyl benzoate is an ester of benzyl alcohol and benzoic acid. It is a non-poisonous, clear, colorless liquid, of neutral reaction. It relaxes tonus or spasm and inhibits the contractions of all smooth muscle organs. It is a valuable medicament in all cases of excessive intestinal peristalsis, and in spasm of the esophagus, stomach, intestine, gall bladder or biliary ducts. It is prescribed in cardiospasm, pylorospasm, diarrhea, dysentery, spastic constipation, enteralgias, enteritis membranacea and in intestinal and biliary colic. The usual dose of the solution is 1 to 2 Cc. (15 to 30 minims) three or more times daily, in water or milk.

Uzara is the native name of a semishrub indigenous to the African sea regions. It has not as yet received a botanical name, but probably belongs to the family of the *Asclepiadæ*. Its medicinal properties reside in the roots. It is used in both acute and chronic forms of diarrhea. The tincture of uzara is a 2-per-cent. solution; dose, 1 Cc. (15 minims) six times daily. The tablets of uzara contain 0.005 Gm. ($\frac{1}{3}$ grain); dose, three to six tablets a day. A combination of uzara and tannin is marketed under the trade name of uzaratin. All these preparations must be used with caution on account of the poisonous properties of uzara.

Astringents and Protectives.—Medicinal substances capable of exerting a favorable influence on the intestinal mucous membrane while it is in a state of inflammation attain this end either by their astringent effect or by depositing a protective covering upon the mucous membrane which acts as a local sedative. Some drugs produce both these effects simultaneously; others exert in addition a disinfectant action on the contents of the intestine.

(a) *Astringents.*—The astringents possess the power of precipitating protein bodies, gelatin and mucus, and thereby forming albuminates. They affect all tissues similarly, but have in addition a special effect on mucous membranes, in contact with which they deposit new solid particles (newly formed albuminous compounds) in the tissue interspaces. This results in a contraction of the tissues. A sort of anemia is produced in the tissues, which thus become poor soil for bacterial growth. The mucous secretion diminishes, and the membrane soon loses its hyperemia; in short, the inflammatory processes decline. The astringent action, however, should be confined to the surface, to form a thin superficial coating, for if it should happen to penetrate more deeply the tissues might be destroyed. It is therefore possible that sub-

stances used in small quantities or in weak solutions, with an astringent effect only, might prove caustic if used in greater concentration.

The astringent best known and most extensively used is tannic acid. Pure tannic acid is not adapted to the treatment of the diseased mucous membrane of the intestine, because it rapidly unites with protein in the stomach, entering the intestine in a combined and inactive form. In the alkaline intestinal secretions these albuminates are converted into alkali-tannates, which are capable of producing only insignificant astringent effects. Moreover, the effect of pure tannic acid on the mucous membrane of the stomach is caustic and may result seriously. For this reason the compounds are used instead of pure tannic acid.

The tannin-albumin compounds are insoluble in acid media, and are therefore not acted upon by the acid gastric juice, but in the alkaline intestinal secretions a sufficient amount of nascent tannin is liberated.

Tannalbumin, a compound of this class, is prepared by heating tannin with albumin; it contains 50 per cent. of tannin. The beneficial effect of this compound in diarrhea is recognized. The dose for adults is 1 Gm. (15 grains), for children 0.5 Gm. (7 grains), several times daily.

Tannocol (tanningelatin), honthin (keratinized albumin-tannate) and glutanol (a compound of aleuronat and tannin) are administered in similar doses.

A preparation of more recent introduction is tannyl, a compound of oxychlorascin with tannic acid. This is a brownish-yellow powder, odorless and tasteless, insoluble in water and acid gastric juice, having well-marked astringent and antiseptic properties. Tannyl is valuable in catarrh of both the large and the small intestine. It is particularly useful in obstinate cases, and is therefore prescribed in gastrogenic diarrhea and ulcerative processes. Tannyl can be given as a powder or in tablet form. The dose of the powder is 0.6 Gm. (10 grains) or more, three times daily, in cocoa or rice-soup. The tablets should be well broken up before being taken; two tablets may be taken three or four times a day.

Milksomatose occupies a unique position among the tannalbuminous compounds, because of its solubility in water. It is a compound of milk casein and 5 per cent. of tannic acid; a yellow-brown powder, odorless and nearly tasteless. The dose for adults is three tablespoonfuls or more several times a day; for children, one to three teaspoonfuls. Soup, bouillon, milk, claret or tea can be used as a vehicle. Because of its solubility, milksomatose is well borne; it has a distinctly astringent effect. (See page 192.)

Tannugen, or diacetyltannin, has proved to be a useful astringent in both acute and chronic catarrh of the intestine. The adult dose is 0.5 to 1 Gm. (7 to 15 grains), the dose for children 0.25 to 0.3 Gm. (4 to 5 grains), several times daily.

Preparations having similar actions are: Tannoform, a condensation product of tannic acid and formaldehyd, given in doses of 0.3 to 0.5 Gm. (5 to 7 grains); and tannopin, a compound of tannin and hexamethylamin, dose 0.5 to 1 Gm. (7 to 15 grains) three or four times a day. These two preparations are useful as antiseptics also. Tannigen is the least soluble in the gastric juice, tannopin and tannoform more soluble, and tannalbin the most freely soluble of the four. Tannoform is most easily soluble in the intestine, followed in order by tannigen, tannalbin, and tannopin.

Agar can be impregnated with various drugs, and on administration convey their therapeutic properties to all parts of the intestine. Agar-tannin is such a combination, useful in all cases of chronic diarrhea. I have used it, following the suggestion of Einhorn, with gratifying results. The dose is a teaspoonful three times a day. (See page 287.)

A number of vegetable drugs are employed because of the tannic acid they contain. Gambir has taken the place of the catechu of the Pharmacopœia of 1890. It is an extract prepared from the leaves and twigs of *Ouroparia Gambir*, and contains a large percentage of tannic acid and its compounds. Gambir was introduced on account of the difficulty of obtaining in the market true *acacia catechu*. The *Tinctura Catechu Compositæ* and the *Trochisci Catechu* (U. S. P. 1890) are replaced by *Tinctura Gambir Compositæ* (average dose, 4 Cc.) and *Trochisci Gambir*, U. S. P. 1910 (average dose, 1 Gm.).

Krameria has similar astringent properties; it is administered as an extract, 0.3 to 0.6 Gm. (5 to 10 grains); fluidextract, 0.3 to 2 Cc. (5 to 30 minims); or tincture, 2 to 8 Cc. (3ss-ij).

Tincture of kino is prescribed in the dose of 1 to 8 Cc. (15 to 120 minims).

Tannic acid is a constituent of *salvia* (leaves) and *Quercus alba* (bark and fruit—infusion of the former and preparations of the latter as acorn correa and acorn coffee).

Hematoxylon (logwood) has been used for a long time as an astringent, and is well borne by the stomach even when its use is prolonged. It may be given as the extract, 0.3 to 1 Gm. (5 to 15 grains) several times daily in pills or in claret. Recently a fine, brick-red powder has been made by the action of formaldehyd on hematoxylon, which is called almatein. It is particularly recommended for the treatment of tubercular intestinal diseases. It is given in capsules or as pastilles in doses of 0.5 to 1 Gm. (7 to 15 grains) four to six times a day.

Ipecacuanha root has a feeble astringent effect.

The tannin contained in all the above-mentioned preparations has only a slight astringent effect on the gastric mucous membrane, because the tannic acid is liberated very slowly from the drug.

The extracts are preferable to the tinctures because they are less irritating to the gastric mucous membrane.

The employment of whortleberries and blackberries in the diet of intestinal diseases is based on the large amount of tannic acid they contain.

The salts of some metals exert a similar but milder astringent effect upon the mucous membrane of the intestine. This is especially true of the compounds of bismuth. The physiologic action of bismuth on the intestinal mucous membrane is believed to be as follows: the compounds of this metal unite chemically with the hydrogen sulphid that is freely evolved during the processes of decomposition, and are precipitated as an indifferent sulphid of bismuth upon the inflamed or ulcerated mucous membrane, thus forming a protective coating. The bismuth preparations at the same time act as antiseptics (see page 265).

Silver nitrate, protargol and other salts of silver are not now employed internally in the treatment of intestinal diseases.

Lactic acid has been recommended, particularly in tuberculosis.

(b) *Protectives*.—Protectives have the property of covering the inflamed or ulcerated intestinal mucous membrane with a thin protective layer, in a manner similar to that ascribed to bismuth in ulcer of the stomach. Among these remedies are the calcium preparations, *calcei carbonas præcipitatus*, *calcei phosphas præcipitatus*, *calcei salicylas*, and *creta preparata*. These drugs have well-marked astringent and antiseptic properties; they are given as powders, either alone or combined in equal parts, in teaspoonful doses, three times daily. Recently clay (*bolus alba*) has been used in acute and chronic diarrhea, and it seems to act well as a protective in such cases. In acute enteritis it has been given suspended in water, in doses of 60 to 100 Gm. (3ij-ijj). Talcum may be employed in a similar manner; large doses of it, however, are necessary—60 to 100 Gm. at each administration.

Kaolin (*bolus alba*, porcelain clay) and talcum (talc) have a slightly astringent action, but they are more useful as protectives. The virtue of kaolin depends upon its power of removing bacteria and toxins from mucous membranes by mechanical absorption.

Antiseptics and Antifermentatives.—Abnormal processes of putrefaction and fermentation in the intestine are apt to continue and to keep up the inflammatory changes of the intestinal mucous membrane; they may also increase peristalsis, and give rise to pains and other disagreeable sensations through the formation of gases which distend the abdomen (meteorism). The diseased intestinal mucous membrane is therefore benefited indirectly when it is possible to check or to diminish such decomposition processes. This can be accomplished up to a certain degree by the antifermentative and antiseptic drugs. To this class belong

the already mentioned drugs tannoform, tannopin and tannyl, salicylate of bismuth, and calcium salicylate. The antifermentative effect of salicylic acid, it will be remembered, was mentioned when discussing the effect of salicylic milk (see page 176). It may be said that the salicylic preparations are the best antifermentative agents for the intestine. Apart from those referred to, attention may be drawn to magnesium salicylate in doses of 1 to 2 Gm. (15 to 30 grains) three times daily. It acts particularly well in gaseous fermentation, even in the absence of diarrhea, and is not as constipating as bismuth salicylate. Bismuth bisalicylate (gastrosan) may be given in these cases. Salol and acetylsalicylic acid, 0.5 to 1 Gm. (7½ to 15 grains) several times a day, may occasionally be used with good results in intestinal diseases.

Calomel was formerly considered the best intestinal antiseptic. It was supposed that in the stomach the calomel was converted into an albuminate which, passing onward to the alkaline pancreatic juice, was there partially oxidized into corrosive sublimate in the presence of sodium chlorid. Other writers, however, hold that the only antiseptic effect of calomel is that which results from its mild, non-irritating purgative action, as in the case of any other mild purgative. Thus it may be termed an antiseptic by virtue of its stimulating effect upon the transudation of liquid into the lumen of the gut—the bacteria being washed out of the mucous membrane. However, since Strasburger demonstrated an actual increase in the number of intestinal bacteria during the administration of calomel this drug has not been considered of much value as an antiseptic. On the other hand, recent researches in Schmidt's clinic make it appear that calomel does not always increase the number of bacteria, but very frequently reduces it.

Magnesium peroxid acts as an antiseptic by the liberation of oxygen, and is in effect probably related to peroxid of hydrogen. Peroxid of hydrogen acts in the small intestine as it does in the stomach. It increases the natural secretions of the intestinal mucous membrane, and thus increases the natural antiseptic power of the parts. Moreover, it literally flushes the bacteria out of the mucous membrane. Perhaps, in addition, it has a directly germicidal effect on the bacteria in consequence of the liberation of oxygen. Pure peroxid of hydrogen is promptly decomposed both in the air and in the stomach. Schmidt recommends impregnating pure agar with it, this vehicle having the property of giving off the peroxid slowly, so that it passes through the stomach into the intestine without decomposition. In the intestine it is decomposed instantly; peroxid agar, or "oxygar," is therefore available in cases of disease of the small intestine in which the lesion is situated high up, but in the lower parts of the intestine no effect can reasonably be expected from this preparation. It is adminis-

tered in the dose of 1 Gm. (15 grains) three times a day in a wafer or in a little water.

Pure betanaphthol is rarely used. Boas prescribes bismuth betanaphthol quite frequently. Rodari warmly recommends benzonaphthol. This preparation exerts its effect directly in the intestine; it passes through the stomach unchanged and breaks up in the intestine, giving off free naphthol. It does not irritate the intestine. It is given in the dose of 2 to 4 Gm. (3ss j) a day.

Ichthyol is said to prevent decomposition quite well. It is given in pills, 0.1 Gm. (2 grains) every two hours. It may be given as an albuminous compound—ichthalbin, which has the advantage of being tasteless and insoluble in acid gastric juice. The dose for children is 0.1 to 0.3 Gm. (2 to 5 grains) and for adults 2 Gm. (30 grains) several times a day. The administration of ichthyol is occasionally followed by slight gastric discomfort and eructation.

Other remedies are creosote and its derivatives. Creosote itself is given in pills or capsules of 0.05 Gm. (1 grain) four or five times a day. Pills with enteric coating, rendering them insoluble in the stomach, are available. A good substitute for this preparation is guaiacol carbonate; but proposote, a compound of creosote and phenylpropionic acid, is better. Thiocol has been recently recommended. It inhibits peristalsis, and decreases hypersecretion and the number of bacteria; does not irritate the stomach; and is given in doses of 0.5 Gm. (8 grains) three times a day, preferably in the form of tablets. Enterol, a cresol preparation; stryacol, an acid ester; and nosophen (tetraiodophenolphthalein) are sometimes used. Iodoform is of some value in certain intestinal diseases (dysentery). Thyol in rather large doses—4 to 6 Gm. (3j-iss) daily—in capsules, acts as an antiseptic. Menthol may be given in the dose of 0.1 to 1 Gm. (2 to 15 grains) several times a day, in capsules. Resorcinol may likewise be employed in the same manner as in gastric diseases. Saccharin in full doses acts as an antifermentative. A pure culture of lactic acid bacilli (Metchnikoff) has been used; its antiseptic effects are fully described in discussing yoghurt (see page 164).

Yeast also has much power of destroying the toxicity of pathogenic bacteria. The yeast of beer is frequently used for the purpose of disinfecting the intestinal tract.

The first principle in the treatment of intestinal diseases must always be to obtain full control over the pathologic condition by purely dietetic measures, long continued rest, and avoidance of all irritation. These measures failing, it is advisable to assist a regulated diet by the giving of astringent and antifermentative remedies. Astringents are contra-indicated in those cases in which inflammation and diarrhea have arisen from decomposition; the removal of the decomposing contents is the rational treatment.

PURGATIVES.

The effect of purgative remedies depends upon stimulation of the peristaltic movements of the intestine, and partly upon liquefaction of the stools. The latter is brought about by an increase in the natural secretion of the mucous membrane of the intestine, from the irritating effect of the purgative agent, or by an increased transudation of serous fluid into the lumen of the intestinal canal. The increase in peristalsis is brought about by local stimulation of the mucous membrane and its nerves, causing a reflex stimulation of the motor ganglia of the intestine. Some drugs act in this manner on the whole intestinal canal, while others act only on the large intestine. This depends on the comparative facility or difficulty of absorption of the medicaments; the more resistance there is to absorption, the more general will be the peristaltic effect. All purgatives stimulate peristalsis; many, in addition, increase the amount of liquid in the intestine. This fact has been controverted up to the very present. Attempts have been made to explain the liquefaction of the stools by saying that the absorption of water from the bowel is diminished by the purgative and the accelerated peristalsis. Later researches have, however, shown that this does not play any very conspicuous rôle. Adolf Schmidt insists that, as a rule, the diarrhea is referable to the secretion of an albuminous decomposable fluid within the lumen of the gut—"No diarrhea with increased peristalsis without secretion of a decomposable fluid by the intestinal membranes." Thus an explanation is offered for the strongly pronounced decomposition processes which are found in diarrhetic stools. This increase of intestinal fluids is caused by a great variety of agencies—bacterial irritation, thermic and chemical stimuli, coarse foods ill digested in the stomach (*e. g.*, raw connective tissue), and similarly by a large number of purgatives.

A general distinction is made between aperients, which affect only the peristaltic movements and render defecation normal both as to frequency and consistency; laxatives, which induce semisolid or liquid stools; and the drastics which cause profuse watery diarrhea and intense inflammatory irritation of the mucous membrane. The latter need not be considered from a therapeutic standpoint. This classification possesses but little practical value, inasmuch as the aperients become laxatives and the laxatives cathartics when given in large doses, and *vice versa*. It is the dose only which makes the difference. The occurrence of pains, often of a colicky nature, after each evacuation, results from the violently stimulated peristalsis and the irritation of the sensory nerves of the intestine.

Indications for the Administration of Purgatives.—Conditions of acute constipation require a purgative unless they are caused by

inflammatory changes or by stenosis of the intestine. Should any doubt exist regarding these latter conditions, it is better to give an enema. When, however, a purgative is indicated, one ought to be selected which acts promptly and surely, and a large dose should be given. When the stomach is diseased, calomel is the best drug; and when the stomach is normal, castor oil. It must not be forgotten that purgatives may cause great disturbance to the gastric secretion. The administration of any purgative ought to be discontinued as soon as possible. In cases of chronic intestinal stasis, purgatives are unavoidable and act more surely than enemata, but only mild drugs should be employed, that irritation of the diseased intestine may be avoided. Purgatives are indicated in those cases of atonic constipation only in which it has been found impossible to obtain results by means of dietetic and physiotherapeutic methods. They should be used in cases of acute intestinal catarrh in order to bring about as rapidly as possible an evacuation of the decomposed and disintegrating fecal masses. Purgatives in such cases act literally as antiseptics, inasmuch as they wash out the bacteria from the mucous membrane by stimulating the normal glandular secretion. Calomel and phenolphthalein are particularly valuable in these conditions. When the bowel is completely occluded, the use of enemata is, of course, the only proper treatment. Purgatives should not be given in the atonic constipation of youthful patients; and they are absolutely contra-indicated in spastic constipation. It is a distinct therapeutic error to treat constipation in cases of chronic enteritis by means of purgatives.

Certain drugs which, though they have no specific purgative action, assist in the passage of feces, should be mentioned here. Opium must be placed first in this respect, as being the most important. Its action in placing the bowels at rest and thus causing the cessation of cramps has already been described. Opium relaxes the intestinal muscle fibers and thereby opens the way for the free passage of fecal matter in cases of spastic constipation. Atropin acts very similarly; its action, however, is not so decided as that of opium. In large doses it has a paralyzing effect upon the nerve endings. Atropin may therefore be combined with opium. Atropin sulphate is given subcutaneously and in pills or tablets; maximum single dose, 0.001 Gm. ($\frac{1}{60}$ grain). The extract of belladonna may be given in doses of 0.015 Gm. ($\frac{1}{4}$ grain); when used for any length of time, caution is advisable, lest poisoning result. Atropin may be replaced by eumydrin, twenty drops of a 1:1000 solution three times daily. Physostigmin (eserin) has a stimulating effect on peristalsis; it directly influences the contractile muscles. Though employed largely in veterinary medicine, it is always somewhat dangerous in human practice and should be used only in extreme cases. In severe tympanites, ileus, meteorism,

and paralysis of the intestine after operations, physostigmin is sometimes prescribed; in such cases it is given in the form of physostigmin salicylate in doses of 0.0006 to 0.002 Gm. ($\frac{1}{1000}$ to $\frac{1}{500}$ grain). There is nothing more reliable, however, for the prevention or relief of postoperative intestinal paresis than pituitrin (pituitary extract), administered hypodermically or intravenously; the dose is 1 to 2 Cc. of the standard preparation, or half this quantity of pituitrin "surgical." Strychnin stimulates peristalsis and is given to adults, especially after operations, in doses of 0.003 Gm. ($\frac{1}{200}$ grain), being preceded by a saline purgative. For the relief of spasm, benzyl benzoate in doses of 2 Cc. (30 minims) may be given with any of the above named preparations.

The number of drugs used as purgatives proper is extremely large. The mildest purgatives are the fruit acids, especially when they are taken as slowly absorbable acid salts (tartrates, citrates) or in combination with colloid materials (pectin, vegetable mucus) and mild laxative carbohydrates (sugar). A large number of fruits (as apples and grapes) contain a mixture of these materials and for this reason play an important role in the dietetic treatment of constipation. Grape juice contains acid potassium tartrate. Some kinds of jam (pulpæ) occupy an intermediary position between foods and purgatives; *e. g.*, prune jam (pulpa prunorum), elderberry jam (pulpa sambuci), quince jam (pulpa cassia fructus), and tamarind jam (pulpa tamarindorum depurata), and the marrow of the skins of *Tamarindus indica* made into a confection. The latter contains a mixture of citric, malic, and tartaric acids, and acts as a purgative in doses of 15 to 30 Gm. (3ss-j). The confection *sennæ* consists of powdered senna, tamarind, fig, oil of coriander, cassia, prune, and sugar, neutralized by means of carbonate of magnesium; dose, 4 to 8 Gm. (3j ij). Manna belongs to this group; it is the dried juice of *Fraxinus ornus* (habitat South Italy), containing 60 to 80 per cent. of the active principle mannite. Manna in doses of 15 to 60 Gm. (3ss-ij) acts as a mild purgative, but is not used as much as formerly. Of the carbohydrates, various kinds of sugar (cane-sugar, grape-sugar, and milk-sugar) act as mild purgatives. The latter two are somewhat more active than the first mentioned. The mild purgative effect of honey is due to the presence of these sugars.

The various substances mentioned above belong to the aperient group. Those now to be considered are usually classed among laxatives. A few inorganic salts may first be mentioned, which are especially represented by sodium sulphate (Glauber salt) and magnesium sulphate (Epsom salt). These salts act by contact as slight irritants. Even in small doses they are laxative, because they are capable of passing along the whole length of the intestine, being not easily diffused or absorbed. This is due, partly, to their ability to unite chemically with a large quantity of water. Not

only are they absorbed slowly, but they hinder the absorption of water; moreover, they stimulate intestinal secretion and transudation, causing liquid evacuations. The effect of these salts on digestion has been studied by means of roentgenographic observations. It is seen that the salt solution retards the motility of the stomach but strongly accelerates the passage of the chyme through the small intestine, liquefies the contents of the large intestine, and urges the feces to the sigmoid flexure, so that in a few hours there results a thin, watery stool. These salts are given in doses of 15 to 30 Gm. (one to two tablespoonfuls) dissolved in a glass of warm water; they are best taken in the morning, one hour before breakfast. It is a good plan for the patient to take some exercise, because bodily motion accelerates the passage of the saline solution from the stomach into the intestine and also aids in the onward motion of the fecal mass. If exercise be neglected, it may be that the salts will produce no effect. This is the reason why these laxatives are not prescribed for patients confined to bed. When employed correctly the mineral salts do not cause any pain, but induce a free defecation in from one-half hour to three hours. The same effects are produced by Carlsbad salts because of the Glauber salt they contain, and by all the well-known mineral waters containing Glauber salt. As a rule Glauber salt is preferred to Epsom salt, on account of its better taste. To improve the taste, lemon juice may be added to any of these salt solutions.

To this group of salts belong quite a number of others which have a similar action. Potassium bitartrate (cream of tartar), soluble in water, has an agreeable acidulous taste. It is a mild purgative, combining the saline and acid effects in doses of 2 to 15 Gm. (3ss-iv). Potassium and sodium tartrate (Rochelle salt), readily soluble in water, has a cooling saline taste and is administered in doses of 8 to 30 Gm. (3ij-3j). Sodium phosphate is a mild purgative with a saline taste, and is readily soluble in water; the dose is 0.3 to 3 Gm. (5 to 45 grains). *Liquor magnesii citratis* (solution of magnesium citrate), a solution of magnesium citrate with excess of citric acid and carbon dioxide, is marketed in corked and wired bottles; it effervesces when the bottle is uncorked; dose, 60 to 240 Cc. (3ij-vij).

Sulphur is a mild purgative. It is insoluble in the stomach, where it causes no discomfort. It is dissolved in the alkaline intestinal juice, but so slowly and in such small quantities that the greater part is expelled unaltered. The alkaline sulphur compound formed, however, suffices to stimulate peristalsis along the entire intestinal canal. Sulphur is employed in the form of sulphur sublimatum in doses of 1 to 4 Gm. (15 to 60 grains), and as sulphur precipitatum in the same dose.

Hydrargyri chloridum mite (calomel, chlorid of mercury) is a prompt-acting, mild purgative, and may always be given in inflam-

matory conditions. It passes through the intestinal canal for the most part undecomposed, but during its entire passage small quantities are dissolved, which manifest their activity by their effect on the ganglia. The difficulty of solution and absorption explains the great efficacy of calomel. The small quantities dissolved are converted into the sulphid of mercury, and probably also into the bichlorid. The calomel stool looks green—which is due to its containing biliverdin, the conversion of which into urobilin is prevented by the calomel. The dose of calomel is 0.006 to 0.3 Gm. ($\frac{1}{10}$ grain to 5 grains) several times daily. This mercurial purgative is not suitable for prolonged administration, because of the danger of producing constitutional symptoms.

Oleum ricini (castor oil), the fixed oil (light yellow, viscid) expressed from the seed of *Ricinus communis*, has an action similar to that of calomel, being both mild and effective. It contains the ester of ricinoleic acid, which, when liberated in the gut by the saponifying action of the pancreatic juice, becomes active. Castor oil acts mechanically also, lubricating both the mucous membrane and the fecal mass. It may be employed in inflammatory conditions. The evacuations following its use are painless and semi-solid. The dose is 15 to 30 Gm. (one to two tablespoonfuls). Its nauseating taste makes it difficult of administration to sensitive persons and children; but it may be given in elastic gelatin capsules. Tasteless preparations of castor oil are sold in the shops.

Another group of vegetable purgatives that are freely employed belongs under one heading, because the active principles are either identical or closely related to one another. These are senna, rhubarb, and aloes. The active principles of these plants are derivatives of anthracene which, with sugar, form glucosides. These medicinal agents are further related by the fact that they are active only in the large intestine.

A vegetable cathartic that is frequently employed is podophyllum (May apple, mandrake). It is a yellowish powder which is separated from the alcoholic extract of the root of *Podophyllum peltatum* by the addition of water. The active principle, podophyllin, is a mild purgative; it is prescribed in small doses—0.008 to 0.03 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain).

Jalap and colocynth are drastic purgatives. On account of their irritating properties and the depression following their use they are rarely prescribed.

Phenolphthalein is met with under many commercial names. It is a mild laxative, and induces painless evacuation of the bowel. It is also said to possess some disinfectant action. It strongly stimulates intestinal secretion and sweeps out the bacteria which are settled in the lower folds of the mucous membrane and in the lymphatic channels. Clinical experience proves that phenolphthalein is a valuable purgative. The discovery of this property

was purely accidental. The chemical was being used to color artificial Hungarian¹ wines and thus prevent their substitution for genuine wine. Those who drank the artificial wines suffered from diarrhea, and as soon as they discontinued their use the diarrhea ceased. Phenolphthalein passes through the acid stomach unchanged, but reaching the intestine some of it combines with the alkaline substances, forming a sodium salt. This latter is of low diffusive power and its presence induces a high osmotic pressure resulting in a copious accumulation of fluids. It is said that 85 per cent. of the drug ingested is found in the feces. Its presence can be readily demonstrated by adding an alkali to the feces, when a purple color will quickly develop.

Phenolphthalein causes evacuation of the bowel in about six hours after the dose is taken. Its use is not followed by increased tendency to constipation, as is the case with many other purgatives. It may be given in capsule, powder, or pill. The dose is 0.06 to 0.3 Gm. (1 to 5 grains) once daily or oftener. Recently it has been stated that irritation of the kidneys and abdominal pain occasionally follow the use of phenolphthalein. It is therefore advisable to exercise some caution in its administration.

The special physiologic action of agar and liquid petrolatum will be discussed in the chapter on Chronic Constipation (XXXVII). Agar will absorb fluid medicaments and later gradually liberate them, thus ensuring the application of the medicament to large areas of the intestinal mucosa. To prepare medicated agar, the medicinal agent is dissolved in boiling agar-water, and the solution, thoroughly mixed, evaporated to the original dry agar volume.

The following combinations have been suggested by Einhorn:

Phenolphthalein-agar. Each level teaspoonful equals 0.03 Gm. ($\frac{1}{2}$ grain) phenolphthalein.

Rhubarb-agar. Each teaspoonful equals 1 Cc. (15 minims) fluid-extractum rhei.

Calumba-agar. Each teaspoonful equals 2 Cc. (30 minims) fluid-extractum calumbæ.

Gambir-agar. Each teaspoonful equals 2 Cc. (30 minims) tincture gambir compositæ.

Tannin-agar. Each teaspoonful equals 0.03 Gm. ($\frac{1}{2}$ grain) tannic acid.

Simaruba-agar. Each teaspoonful equals 1 Cc. (15 minims) tinctura simarubæ.

Phenolphthalein-agar and rhubarb-agar can be conveniently used in the different varieties of constipation. One teaspoonful of either in water, twice daily, is the average dose.

Calumba-agar has proved valuable in cases of colitis (appearance of considerable mucus in the stool) with normal defecation. The

¹ Gillette: Journal of the American Medical Association, January 20, 1910.

average dose is one teaspoonful in water, after meals (three times a day).

Gambir-agar, tannin-agar and simaruba-agar are valuable in diarrheal conditions, acute or chronic. The average dose is one teaspoonful three times a day, after meals (see page 277).

Aloin, apocodein, eserine, atropin and eumydrin have been injected subcutaneously. It has been found that this method of administration is not injurious. The subcutaneous injection of magnesium sulphate, cascara sagrada, pituitary extract and hormonal is fully described in Chapter XXXVII—Constipation.

CHAPTER XV.

DISEASES OF THE MOUTH.

THE mouth may be anatomically divided into two principal parts: the anterior or actual cavity, containing the tongue, the organs of taste, the teeth, and the orifices of the salivary glands; and the posterior or bucco-faucial cavity, also called the isthmus faucium, extending from the soft palate to the epiglottis.

For conciseness of terminology the following additional subdivisions have been named: (1) the vestibulum oris, the space between the teeth and the cheeks and lips, demarcated posteriorly by the junction of the main oral cavity and the isthmus faucium; (2) the lingual region, characterized by abundant musculature, peculiar formation of the papilla, the follicular glands, and the terminations of the nerves of taste; (3) the fundus of the mouth, an irregular fissured space demarcated anteriorly by the lower teeth, laterally by the submaxillary bones and their teeth. Underneath the buccal fundus are situated the submaxillary salivary glands, resting upon the geniohyoglossus and mylohyoid muscles. These muscles separate the fundus from the subcutaneous connective tissue of the neck. The transition to the faucial cavity is formed by the tonsillar region, which likewise includes the follicles at the base of the tongue. All the organs of the oral cavity, with the exception of the teeth, are invested with epithelium. The various parts of the mucous membrane differ considerably, according to the quantity of glands and their manner of adhesion to their base. The epithelium everywhere consists of striated pavement cells, and resembles the epithelium of the external skin except as regards firmness, which is normal to the red part of the lips and the dorsal aspect of the tongue only. The epithelial layer is rather thin at the inner labial mucosa, while at the dental margins and on the surface of the tongue it attains to considerable thickness. The vascular supply of the mucosa of the mouth is very abundant. The course of the veins corresponds in a general way to that of the arteries. There is also a dense net of lymph vessels and lymph nodules in the mucous membrane and other structures of the oral cavity.

The fact that the oral cavity is so frequently the seat of pathologic affections is explained by the incessant introduction of micro-organisms by means of food and eating utensils, the fingers and the respired air. The moist heat prevailing in the mouth furnishes

a most favorable condition for their growth, and it is therefore not to be wondered at that an exceedingly large number of many varieties of bacteria are demonstrable in that cavity. As a matter of course, the number and kind of bacteria differ according to the method of examination and the time bestowed upon it, and also upon the care given to the cleansing of the mouth. The various bacteria cannot be readily differentiated, for the reason that many of them cannot be grown at all, or only with great difficulty, on an artificial culture medium. There is no doubt that many of those that cannot be artificially grown are harmless, inasmuch as they are nearly always present in healthy individuals. Besides, there are numerous pathogenic microorganisms in the oral cavity, notably the *Staphylococcus pyogenes aureus* and *albus*, and more frequently even the *Streptococcus pyogenes*. There are also numerous pneumococci. Tubercle bacilli are frequently found in the mouths of tuberculous persons, leprosy bacilli in leprosy individuals, and typhoid bacilli in typhoid cases. These microorganisms may have gained secondary access to the oral cavity. Constitutional infections are not infrequently caused by the microorganisms entering through the mouth and gaining access to the circulation by deglutition, respiration, or direct absorption. It is probable that they can also enter through the epithelial crypts of the tonsils. A simple examination of the tonsil can be made by depressing the tongue with a laryngeal mirror placed well back at its base and pressed against the tonsil at its lower border. By drawing the mirror upward, in this manner milking the tonsil, any infection would become apparent, as pus would show on the surface of the mirror.

Oral Sepsis.—There is a close relationship between oral sepsis, alimentary toxemia, and intestinal stasis. Lane asserts that pyorrhea alveolaris is caused by intestinal stasis (see page 697). Chronic constipation produces alimentary toxemia. The absorption of toxins poisons the whole system, inducing functional disturbances and organic diseases of a more or less serious nature. The oral cavity, providing a fruitful soil, soon becomes infected. On the other hand, pus which has formed around the teeth and gums is carried into the intestinal canal, inducing or aggravating a general septic condition. The whole process is known as intestinal toxemia. The absorption of toxins from the alimentary tract devitalizes the system and lowers its powers of resistance. Both oral sepsis and intestinal stasis must be corrected in order to bring about recovery in many obscure diseases (see page 689).

Focal Infection.—Hygiene of the mouth is one of the most important considerations in the treatment of diseases of the digestive organs. Experimenters have been able to induce in the lower animals gastric and duodenal ulcer, cholecystitis, pancreatitis, appendicitis, neuritis, oöphoritis, rheumatism, arthritis deformans,

PLATE XXIII



Focal Infection.
Apical abscesses and gum-boil

ease will prove to be preventable, or amenable to proper treatment when brought to the notice of the physician. In other words, progressive ill-health from apparently unexplainable causes may be prevented or cured by the removal of chronic foci anywhere in the body, and the full attention and energy of the physician should be directed to their discovery. A systemic infection from unrecognized, unsuspected or unremoved chronic foci may continue for years, gradually poisoning the system. Even if one infected focus has been discovered and removed, and the systemic disease does not clear up, it does not follow that the theory is wrong; in all probability there is yet another focus of infection which will have to be searched for and removed to ensure success.

Any part of the body may harbor a focal infection; and the recognition of the fact that these infections are responsible for many diseases which the profession has not been able to deal with satisfactorily on the principles of symptomatology, has gone far toward arresting professional attention and directing it on the right path in its search for etiologic factors. Such centers of focal infection are found in cholecystitis, appendicitis, submucous abscesses, salpingitis, vesiculitis seminalis, and prostatitis; but the one site to which the vast majority of all investigation has so far been directed as the principal offender is in the head, in and about the tonsils, the roots of the teeth, and the accessory sinuses. (See Plate XXIII.)

Now we have it on the authority of all the authors who have concerned themselves with the investigation of this question, that the acutely inflamed tonsil, where the crypts are full of dead cells, blood and dust particles, and countless bacteria, may be the focus from which may arise otitis media, sinusitis, mastoiditis, bronchitis, pneumonia, gastric and duodenal ulcer, endocarditis, myocarditis, pericarditis, cholecystitis, appendicitis, iridocyclitis, arthritis, rheumatic fever, and perhaps other diseases. The chronically inflamed tonsil with pouting crypts may, besides, lead to nephritis and interstitial hepatitis.

The variations in the strepto-pneumococcus and other groups, while first discovered in cultures grown in the laboratory, are apparently also present in focal infections, the tissues serving as a culture medium. Blood supply, oxygen tension, and unknown biochemic or other factors modify or entirely change the bacterial characteristics; this is one explanation of the development of arthritis as a consequence of tonsillitis, of endocarditis from the presence of the *Streptococcus viridans* or hemolyticus in alveolar abscesses, and of similar affections on the same principle.

The *Streptococcus viridans* or hemolyticus, to which reference has just been made, has a special pathogenicity for malignant endocarditis, with a predilection for old valvular scars and the endocardium, where it causes the development of enormous vegetations and thrombus formations. The only hope for the patient in

these cases is complete eradication of the original focal infection whence the germ is carried into the circulation. Myositis, arthritis, and other chronic diseases are instances in which the same micro-organism exerts its baneful influence in milder degrees.

The specificity of the strepto-pneumococcus group was beyond the pale of understanding prior to the remarkable achievements of Rosenow, who proved the transmutability of these organisms. An important factor in this transmutation seems to consist in the oxygen supply of the tissues, so that characteristics may develop which render the organism pathogenically specific in the myocardium, endocardium, pericardium, gall bladder, pancreas, kidney, mucous membranes of the stomach and intestine, tendons and aponeuroses.

The similarity of the pathogenic organisms in the original focus and in the remote infected tissues may be regarded as proving the etiologic relation between the two, for many bacteria retain for a long time the peculiar properties which determine their characteristic localization. The idea readily suggests itself that other diseases, whose etiology is still obscure, may have a similar origin, and there is consequently a wide field open for further research work, experimentation and discovery.

The mouth should be kept free from infection at all costs, even though it should mean the removal of every tooth in the head, for a toothless mouth is preferable to one containing a single focus that menaces the health of the patient. Frequent roentgenograms should be taken, not only to discover every suspicious root, but also to inspect from time to time all crown and bridge work, in order to discover any disease in the hidden parts. There is no greater menace to health than crowned and bridged teeth, to say nothing of imperfectly filled and dead teeth, inasmuch as the hidden bacteria are preserved thereby and are ever ready for mischief. No dentist should devitalize a tooth, or attempt to fill the roots of a devitalized tooth which is to be preserved, without the aid of roentgenograms. (See Plate XXIII.)

Constitutional diseases due to focal sepsis cannot be successfully treated until the focus of infection is eliminated. Hence the necessity for thorough exploration of the mouth—for the susceptibility of the teeth to decay, of the root sockets to ulceration, and of the tonsils to bacterial attacks, especially streptococcic, makes it probable that, when other sources of infection are unknown, the focus is in the mouth. Especially should chronic focal sepsis without pronounced local manifestations be looked for, since its metastatic consequences are most serious.

GENERAL TREATMENT OF THE DISEASES OF THE MOUTH.

Prophylaxis is of the greatest importance in these diseases, as in all others. Numerous affections of the mouth are preventable,

provided intelligent care be taken to that end. Any special care of the mouth of nurslings before the eruption of teeth is detrimental, as long as the mouth is in a healthy condition. After the teeth appear, the infantile mouth requires the same care as that of the adult.

As a general rule it will be sufficient to brush the teeth carefully at least once a day with a brush and a suitable tooth powder, paste or soap; and to mechanically remove food remnants by rinsing after each meal if possible, but in any case several times a day. The brush should be applied with an up-and-down motion, for the double reason that it then operates best to remove food particles and does not tend to cause erosion of the neck of the tooth. It should be applied inside as well as outside of the denture. The following tooth powders and pastes can be recommended:

	Gm. or Cc.	
R—Calcii carbonatis præcipitati	90 0	℥ij
Saponis		
Camphoræ	℥ss 6 0	℥iss
Olei menthuæ piperitæ	1 0	℥xv
Misce et ft. pulv. subtilis.		

	Gm. or Cc.	
R—Calcii carbonatis præcipitati	30 0	℥j
Pulveris iridis		
Pulveris calami	℥ss 10 0	℥iss
Pulveris myrrhæ	5 0	℥iss
Olei rosæ	3	℥v
Misce et ft. pulv. subtilis.		

	Gm. or Cc.	
R—Calcii carbonatis præcipitati	60 0	℥ij
Pulveris iridis	20 0	℥v
Saccharini	0 01	gr. 1
Sodi bicarbonatis		
Magnesi oxid.	℥ss 4 0	℥j
Misce et ft. pulv. subtilis.		

	Gm. or Cc.	
R—Calcii carbonatis præcipitati	60 0	℥ij
Acidi borici	40 0	℥iss
Saponis	40 0	℥iss
Saccharini	0 01	gr. 1
Aquæ ammoniæ	1 0	℥xv
Olei menthuæ piperitæ	1 3	℥xx
Olei rosæ	1	℥ij
Glycerini, q. s.		
Misce et ft. pasta.		

Carious teeth must be repaired, being veritable hotbeds for the growth of microorganisms. Mechanical cleansing of the teeth is much more important for the purpose of removing bacteria from the mouth than the application of powerful antiseptics. The brush should be applied to the teeth, including the inner and masticating surfaces, both transversely and vertically. The removal of food remnants from between the teeth requires the use of dental floss. Toothpicks should not be used, for they are incom-

patible with the requirements of a rational hygiene of the mouth, whether they are made of wood, metal, or any other material. The habit of brushing the surface of the tongue is to be deprecated, since it may lead to atrophy, fissure, or fragility of the epithelium. Individuals wearing dental and palatal prostheses should subject these to very careful mechanical cleansing, and remove them overnight. They should also exercise more than usual care in maintaining a healthy condition of the mouth.

Mouth-washes have no antiseptic properties, unless they contain such drugs as menthol, permanganate of potassium, hydrogen peroxid, thymol, or salol. A saturated solution of sodium silico-fluorid makes a very effective mouth-wash in oral infections. The following solutions can be recommended: boric acid (3 per cent.), thymol (0.05 per cent.), or hydrogen peroxid (1 per cent.).

	Gm. or Co.	
R—Potassen permanganatis	1 0	gr. xv
Aque destillatæ ad	50 0	3 iss

Macer.

Sig.—Add drop by drop (5 to 10 drops) to a glass of water until the solution is pink or light violet.

	Gm. or Co.	
R—Thymolis	1 0	gr. xv
Acid. benzoici	6 0	3 iss
Tincturæ eucalypti	24 0	3 vj
Aque destillatæ q. s. ad	1500 0	Onj

Macer.

Sig.—May be diluted, and used as a mouth-wash.

Aside from the antiseptic effect, an astringent and refreshing action is often desirable. This is accomplished by aluminum acetate, or by tincture of myrrh, rhatany, or nutgall.

	Gm. or Co.	
R—Liquoris alumin. acetatis	60	3 ij

Sig.—Thirty drops to a glass of water.

	Gm. or Co.	
R—Tincturæ myrrhr.		
Tincturæ krameris aa	7 5	3 ij

Macer.

Sig.—Thirty drops to a glass of water.

Persons whose occupation predisposes to mouth diseases should exercise great caution on this account. Occupations of this kind are those in which mercury, phosphorus, arsenic or lead is used. Minute or coarse particles of these poisons are apt to enter the mouth, in the work rooms or by contact with fingers soiled during work. The obvious prophylaxis is adequate supervision and instruction. It is equally clear that careful hygiene of the mouth is of the greatest prophylactic importance, and work-people with carious teeth should never be employed in any of these occupations. It is well known, for instance, that phosphorus necrosis never

occurs except in the presence of caries of the teeth. Other occupations facilitate the spread of infectious diseases by the mouth, an instance of which is furnished by the frequent spreading of tuberculosis and syphilis among glass-blowers.

Scrupulous oral hygiene is doubly indicated in the presence of any affection, local or constitutional, which, not originating in the mouth, nevertheless involves the mouth. In febrile affections, such as typhoid, pneumonia, or grave septic diseases, changes in the mucosa of the mouth are often occasioned by swelling and loosening of the gums, owing to heavy coating of the tongue and disturbance of the general condition. Patients should be required to rinse their mouths and brush their teeth as soon as they are able to do so. Similar measures are indicated in grave affections of the brain and spinal cord, and in those leading to severe constitutional disorders of nutrition (diabetes, leukemia, nephritis, cardiac disorders, etc.).

The rules of prophylaxis, however, will not avail when the oral cavity has already become distinctly affected. The most important thing then is to keep any possible detrimental influence away from the affected areas. Tooth-brushes, frequently sterilized, preferably by repeated rinsing in hot soap-water, should be used with the greatest caution; they should not be applied too energetically. Filling or removing carious teeth should be done without delay, in order to avoid mechanical lesions of the mucous membrane. Ill-fitting artificial teeth should not be tolerated. Coarse articles of food, likely to irritate the mucous membrane, should be avoided (figs, nuts, etc.). Highly sugared dishes, which readily lead to acidification, are not permissible. Voluble talking should be forbidden. It goes without saying that smoking, alcoholic beverages and highly spiced dishes should be excluded.

In spite of the desirability of avoiding mechanical lesions, the mouth should be thoroughly cleansed in all serious oral affections. This applies especially when the treatment of the mouth and tongue is hindered by pain or swelling, which preclude the automatic cleansing by the tongue and the mastication of food. In these cases mouth-washes are necessary, less for their disinfecting than for their purely mechanical effect. For this purpose any of the following may be used: Boric acid (3 to 4 per cent. solution), borax (5 per cent.), menthol (10 per cent. alcoholic solution, 10 to 20 drops stirred in a glass of water), thymol (0.1 per cent.), hydrogen peroxid. Superabundance of sputum or mucous secretion may be checked by aluminum acetate (a teaspoonful of a 2-per-cent. solution to a glass of water). Pronounced *fetor ex ore* and the sensation of bad taste associated therewith may often be removed by a solution of potassium permanganate (0.025 to 0.05 per cent.) or hydrogen peroxid (1 per cent.). If at all possible, patients are to rinse their mouths at frequent intervals with one

of these washes. Unfortunately, they are often unable to do so on account of the inflammatory condition of the mouth; it will then be necessary to wash the mouth with the aid of an irrigator or syringe. In circumscribed affections which are not readily accessible, and especially in the presence of dry deposits defying easy removal, it is advisable to paint the affected places with mild concentrated antiseptics. Very effective service is rendered by the boroglycerid mixture and the following combinations:

	Gm. or Cc	
R. Tincturae iodi,		
Tincturae gallie,		
Tincturae myrrhæ	ss 10 0	3iiss
Misce.		
Sig.—For painting the gums, lips and tongue.		

	Gm. or Cc	
R. Tincturae gallie,		
Tincturae myrrhæ,		
Tincturae kramerie	ss 10,0	3iiss
Misce.		
Sig.—For painting the gums, lips and tongue.		

Non-poisonous antiseptic substances may be conveniently administered to sore-mouthed nurslings, by mixing with sugar and wrapping up in some sterile dense covering, the bolus being then given them to suck.

Potassium chlorate may cause methemoglobinemia and methemoglobinuria on account of the fact that in severe affections of the mouth swallowing of the irrigating fluid cannot always be entirely avoided. With this precaution, the use of a saturated solution of potassium chlorate is of great value.

Altogether indispensable in the treatment of buccal affections is iodoform; it is the sovereign remedy in all lesions of the mouth associated with loss of substance, in all operative or traumatic defects, and in ulcerous processes of all kinds.

At the buccal fundus and the vestibulum oris, iodoform is best applied in the form of gauze (10 per cent.). This is inserted in strips, which will remain in position owing to the fact that in severe and painful affections of the mouth the patients do their best to avoid all movements of the tongue and mouth. Thus it will happen that the gauze remains *in situ* undisturbed for several days, during which time the affected part is protected and unmolested by extraneous irritations while at the same time a very effective disinfection is being accomplished. This saves much pain and inconvenience, such as are unavoidably associated with irrigation and painting. However, the gauze will not remain undisturbed at the palatal roof, at the inner surface of the alveolar processes, or at the soft palate. For these localities iodoform powder may be insufflated upon the affected parts, or iodoform mass painted

upon them with a gauze or cotton pad. The edges of the gums may likewise be treated with either powder or mass.

Iodoform mass is prepared in the following way: The quantity of iodoform decided upon is mixed with about 10 parts of a 1:1000 sublimate solution, 2-per-cent. phenol, or 3-per-cent. boric acid, and the mixture allowed to stand for twenty-four hours in a tall graduate or beaker, after which it is decanted and ready for use. Iodoform mass has no unpleasant by-effects except the odor and occasionally slight salivation. It follows that iodoform treatment is advisable, above all, in ulcerations with slimy deposits, severe gangrenous stomatitis, and all lesions of the buccal mucosa. The substitutes for iodoform are not complete representatives.

Cauterization of the mucous membrane and the gums, formerly much resorted to, can usually be dispensed with. Occasionally it is useful in the beginning of severe gingivitis. Cauterizing agents of this description are: Silver nitrate (in the shape of the stick or as a 10- to 20-per-cent. solution), chlorate of zinc solution (8 per cent.), 20-per-cent. chromic acid solution, or chromic acid crystals; lactic acid in solutions of from 80 per cent. to concentrated, especially in tuberculous ulcers. In some cases lactic acid is excelled both in efficacy and painlessness by a pap of iodoform and lactic acid, which is prepared by moistening iodoform powder with a little alcohol and stirring to a thick mass with a solution of lactic acid. Corrosive sublimate may be used as a cauterizer, the following form being particularly suitable for application to mucous plaques:

	Gm. or Co.	
R—Hydrargyri chloridi corrosivi	0 3	gr. v
Ethers,		
Alcoholis.	AA 15 0	3iv
Misce.		
Sig.—Apply with caution.		

A very difficult problem in cases of severe and painful oral affections is sometimes presented in the matter of food supply. When the affected lips and teeth have difficulty in seizing the food, or in trismus, all fluid or semifluid articles of diet have to be administered in a way to reach the base of the tongue. This is accomplished by means of a beaked cup. Should the oral opening be even too narrow to accommodate this vessel, the latter may be provided with a thin rubber tube, by means of which liquids and soup containing finely minced meat can be conveniently conveyed to the base of the tongue. Instead of a beaked cup, a funnel or an irrigator with a rubber tube attached will answer the purpose. When the mouth can be opened a little farther, the necessary quantity of food can be administered with a small spoon. When deglutition is impeded, liquids and liquid foods must be administered very slowly. Patients can often aspirate liquids very well through a

glass or rubber tube. Infants are usually given the ordinary nursing bottle, with a rubber cap, to drink from. Painful deglutition can be relieved by painting or spraying with a 5-per-cent. cocain, eucain, or novocain solution; or one of these substances may be administered as tablets—cocain, 0.005 Gm. ($\frac{1}{4}$ grain); eucain or novocain, 0.01 Gm. ($\frac{1}{2}$ grain). Orthoform and anesthesin may also be administered as powders. Painful ulcerations are covered with a mass prepared from powdered orthoform or anesthesin and glycerin. Should these substances not be sufficiently powerful, or should deglutition be impossible from whatever cause, a pharyngeal tube should be inserted for introducing the food. This may consist of a simple rubber tube or catheter, which need not be inserted farther than just beyond the cricoid cartilage, the food passing through the esophagus into the stomach by gravity. Should even this procedure cause considerable pain, there is nothing left but to introduce the tube into the esophagus through the inferior nasal canal. This is best done through the right nostril, the tube being anointed with oil or petrolatum. When the interior of the nose is much swollen or the mucous membrane so greatly irritated as to cause sneezing, the passage of the tube can be facilitated by a cocain spray; the tube is then rapidly inserted up to the posterior faucial wall, and the patient requested to swallow as it glides down. Entrance of the tube into the glottis can in most cases be prevented. If no cough follows, the tube is sure to have reached the esophagus, and the liquids may be poured in without causing any pain.

ORAL AFFECTIONS IN GENERAL INTOXICATIONS.

Numerous poisons pathologically affect the mouth, either by absorption or by secretion through the salivary and mucous glands, or by both processes combined.

Metals.—Mercury.—Among metals, mercury occupies the first place. Mercurial stomatitis is a characteristic affection resulting from the administration of mercurial medicaments or from the nature of the patient's occupation. Mercury may be conveyed directly or in the form of vapor (from inunction) to the oral mucosa. By whatever means it finds its way into the organism, it is secreted in large quantities through the saliva and can thus give rise to stomatitis. This affection nearly always attacks the teeth first, preferably at and behind the wisdom teeth; otherwise it usually commences in carious teeth and roots. The gums of these defective teeth become relaxed, red and swollen, with considerable salivation. Similar changes appear in the neighboring parts of the mucous membrane and tongue, after which the process spreads to the other parts of the gums and cheeks, with considerable increase in salivation. The edges of the gums exhibit a more or less exten-

sive zone consisting of desquamated epithelium, detritus, and putrefactive bacteria, and covered with a yellowish-gray, slimy, malodorous mass. There is at the same time a characteristic *fetor ex ore* which, in stomatitis, is of a peculiar metallic character. Gradually the gums commence to ulcerate underneath the slimy layer; the ulcers are covered with a thick yellow and greenish lardaceous deposit and are surrounded by an intensely red inflammatory zone. Similar ulcers may appear at the cheeks, lips, and teeth. The tongue may undergo considerable swelling, showing the impression of the teeth. This condition may be of an exceedingly tormenting nature. The ill-smelling, abundant saliva can only with difficulty be swallowed or expectorated, and escapes involuntarily from the open mouth (salivation). Swelling of the surrounding soft parts may cause spasm, rendering ingestion of food almost impossible. The surrounding lymph glands may swell, and there is often fever. If other grave manifestations of a constitutional mercurial intoxication set in (diarrhea, nephritis), an originally quite harmless stomatitis may change into a serious vital affection. Deglutition pneumonia may also occur. The prognosis must therefore be very guarded in serious cases. The diagnosis will offer no difficulties, especially when the physician thinks of the possibility of mercurial poisoning.

Prophylaxis.—Workmen handling mercury should be urgently warned as to the dangerous effects of this metal and the necessity of carefully cleansing their hands, particularly before meals. Absorption of mercurial vapor cannot easily be guarded against. In inunction treatment the anointed parts are to be well covered and not to be touched unnecessarily. Nurses and others attending to the inunction are likewise exposed to the danger of infection.

Treatment.—If stomatitis occurs in the course of inunction treatment, attempts may be made to counteract in part the effect of the mercury by sulphur baths. Should these be contra-indicated owing to the gravity of the syphilitic affection, arsphenamine may be used. If the application of mercury must be continued, the local treatment should be very energetic, consisting of the most scrupulous cleansing of the mouth. In light cases, gargles and mouth-washes will be found sufficient; in severe ones the mouth is rinsed with an irrigator, or the affected parts are painted with a soft cotton swab or hair-brush. Iodoform mass or tamponade with iodoform gauze is likewise indicated in severe cases (see page 298).

Bismuth.—An affection resembling mercurial stomatitis is caused by bismuth, the injurious effects of which have latterly been observed in an increasing number of cases since the practice has obtained of filling up bone cavities with bismuth paste in chronic ulcerations.

The treatment, after the bismuth deposits have been removed, corresponds to that of mercurial stomatitis.

Lead.—Stomatitis is often caused by lead poisoning running a chronic course. As a rule there is relaxation and swelling of the mucous membrane, and a blue-lead fringe at the edges of the gums resulting from deposits of minute particles of lead and sulphur into the gums.

Silver.—Silver poisoning is characterized by argyrosis, involving a spotted, black-brown discoloration of the entire mucous membrane of the mouth, a discoloration which may also appear on the external skin. There is, however, no real stomatitis.

Metalloids.—Chronic phosphorus poisoning is caused by the handling of *yellow phosphorus*. The stomatitis occasioned thereby commences with relaxation and swelling of the mucous membrane, particularly of the gums, and very rapidly spreads to the maxillary bones, especially the lower. Abscesses undermine the mucous membrane and, perforating them, form fistulae. The process may extend over several years, finally leading to extensive destruction of bone.

In the interest of prophylaxis, yellow phosphorus should be banished from match factories. The work-rooms should be well ventilated and the men instructed to pay careful attention to the hygiene of the mouth and teeth as well as to cleanliness of the hands.

Treatment consists in tamponing the abscesses with iodoform gauze and removing the necrotic parts.

Bromids and iodids in small doses occasionally cause loosening of the teeth and swelling of the gums and abundant salivation. There is a peculiar soft, yellowish deposit upon the teeth, accompanied by a characteristic *fetor ex ore*.

Medicinal Exanthems.—The use of a number of medicaments, notably antipyrin, quinin, phenacetin, and acetylsalicylic acid, occasionally leads to efflorescences of the oral mucosa in the form of roundish blisters and yellowish, easily bleeding deposits. There are similar efflorescences upon the skin.

AFFECTIONS OF THE MOUTH IN CONSTITUTIONAL INFECTIOUS DISEASES.

Numerous infectious diseases give rise to characteristic manifestations in the oral mucosa. In *measles*, the so-called Koplik's spots are a well-known manifestation, appearing, before the eruption of the exanthem, at the interior lining of the cheeks and lips as irregular red patches, in the center of which are bluish-white or yellowish-white spots.

In *scarlet fever* the exanthem of the skin is accompanied by deep reddening of the oral mucosa, the latter being very dry. The tongue is covered with a heavy coat, which desquamates in the course of two or three days. Its upper surface, or possibly the entire tongue, is very red, and the papulae are considerably swollen,

presenting the well-known pictures of raspberry or strawberry tongue.

In *rubeola* the exanthem of the skin is sometimes preceded by a small, tender, spotted, pink exanthem of the soft palate and fauces.

Variella is often localized in the mouth, especially at the hard palate, tongue, and gums. As soon as the blebs desquamate, there appears a yellowish-white shallow ulceration, closely resembling stomatitis aphthosa. Consequently errors in the diagnosis are apt to occur unless the exanthem is of a very characteristic nature.

Variola is sometimes accompanied by severe stomatitis and ulcerous disintegration of the vesicles, which may confluence into large foci with an ulcerous coat, causing considerable pain.

It sometimes happens that, following *vaccination*, vaccine pustules appear on the mucous membrane of the mouth, the vesicles changing to shallow ulcers which run a thoroughly benign course.

In *typhoid* and *paratyphoid* the anterior palatal arch occasionally exhibits ulcers which closely resemble mucous plaques and heal within a few days. Similar ulcers may simultaneously appear on the lips, cheeks, dental margins, and lingual frenum.

Foot-and-mouth disease, according to recent investigations, can infect man, causing blebs and ulcers in the mouth. For prophylactic purposes it is important to see to it that milk from animals suffering from this disease is not used for alimentation.

In *influenza*, stomatitis and herpoid eruptions are of frequent occurrence.

DISEASES OF THE MOUTH IN NON-INFECTIOUS CONSTITUTIONAL DISEASES.

In *hemophilia* there are occasional hemorrhages into the gums. There may also be hemorrhages into the tongue, causing considerable swelling in some parts which have to be incised in order to evacuate the blood.

Chlorosis sometimes gives rise to painful rhagades and excoriations at the angles of the mouth and the dental margins. Changes of this kind are very frequent in pernicious anemia.

Hemorrhages of the gums are of frequent occurrence in *purpura*.

Pronounced stomatitis occurs in *scorbutus*. It is not yet definitely decided whether this disease, as well as *purpura*, is to be regarded as infectious. The first symptom is inflammation of the gums, which become swollen and tender. Gradually the edges become necrotic and detach themselves in shreds. The disease is usually confined to the gums, but it never occurs in nurslings or the aged. There is considerable pallor of the mucous membrane, and exaggerated salivation. The prophylaxis consists of rational oral hygiene. Stomatitis, when present, is treated with mouth-washes, and disappears under appropriate diet and care.

Infantile scurvy (Barlow's disease) is likewise associated with stomatitis, swelling and bleeding of the gums, but without necrosis. Here, again, general treatment and diet will rapidly effect a cure.

In pregnancy, light gingivitis is of frequent occurrence.

Diabetes mellitus and *tuberculosis* are often associated with pyorrhea alveolaris. Stomatitis also occurs in *gouty individuals*.

EROSIONS AND BURNS OF THE MOUTH.

Erosions.—Erosions of the mouth are occasioned by the use of highly concentrated mouth-washes or by swallowing caustic poisons.

Erosions due to mouth-washes occur comparatively often. The sensitiveness of the oral mucosa toward antiseptic mouth-washes differs in different individuals, and in the pathologic mouth is increased, with the result that mouth-washes which normally are well tolerated may lead to erosions. These will form white scabs with reddened and thickened areola, which usually heal within a few days. These scabs are found for the most part at the margin of the tongue and epiglottis, and at the palatal arches and uvula. Unless the possibility of erosions due to mouth-washes is borne in mind, these manifestations may give rise to mistaken diagnosis, as for instance of diphtheria or syphilis. For the purposes of a differential diagnosis it should be remembered that in erosions from mouth-washes the vallecule are seldom involved, whereas the diphtheritic process spreads to these structures.

Caustic poisons, such as concentrated alkali solutions, lye, concentrated soda or potash solutions, aqua ammonia and the acids, cause much severer lesions. Among the acids, sulphuric, hydrochloric, phenol and lysol are the principal offenders. The erosions caused by all these substances present the same general character. The scabs are at first pure white and more or less protruding, and may spread over a large area of the oral cavity. The parts most prominently affected are the labial margins, tongue, and soft palate. The labial scabs gradually dry up, forming brown and black crusts. The scabs of the mucous membrane are gradually desquamated, leaving painful gray-yellow ulcers behind, the borders of which are more or less reddened and swollen. If extensive areas in the vicinity are considerably swollen, and the inflammatory edema continues spreading, the involvement of the entrance to the glottis may cause considerable dyspnea and grave danger to life.

Treatment.—In the first place, the poison has to be counteracted: acids by alkalis, alkalis by acids. To alleviate the pain, small pieces of ice are administered, the mucous membrane is painted with boroglycerid, and an anodyne is given. Food is withheld as far as possible, and artificial feeding resorted to if necessary. Ulcerous points are treated with iodoform.

Burns.—Though the mucous membrane of the mouth has a comparatively high power of resistance to heat, it may be burned by hot liquids, hot potatoes, etc. Severe scalding may result from inhaling hot steam, as in explosions. High degrees of heat produce blisters which soon burst, also thin fibrinous deposits, and swelling and reddening of the mucous membrane. The pains may be so severe as to completely prevent all movements of the mouth and tongue.

LESIONS OF THE ORAL MUCOSA.

Traumatic Lesions.—Traumatic lesions of the oral mucosa are due to extraneous causes and to injudicious biting movements. The lips are often injured, contused and lacerated, by a fall. The gums are exposed to numerous injuries from without and within. Injury to the tongue is inflicted by the teeth in biting; in the case of a fall, pieces of the teeth may break off and stick in the tongue; or foreign bodies (fish bones, splinters of bones) may penetrate the tongue, where they are arrested and cause ulceration. Injuries of the hard and soft palate are often due to a fall with the mouth open. The malar mucosa is often injured by the teeth in mastication.

Treatment.—Lesions in the oral mucosa have a tendency to heal smoothly. Nearly all of them can be sutured, with the exception of those of the fundus. Suturing is at the same time the best means of arresting hemorrhages, which are often severe. When a wound has been exposed to infection, the surfaces should be rubbed with iodoform powder prior to suturing. When the wound is situated low down, and particularly on the floor of the mouth, tamponade with iodoform gauze is indicated.

Thickening of Epithelium.—Glass-blowers sometimes suffer from a considerable thickening of the epithelium of the malar mucosa without inflammatory manifestations—a form of leukoplakia. Old glass-blowers often have their incisors considerably worn down by the action of the mouth-piece of the blow instrument.

Palatal Ulcers.—In the newborn, palatal ulcers are not infrequent. They occur either in the center of the hard palate or in places where the mucous membrane of the palate is elevated by the hamuli pterygoidei of the sphenoid. They are found only in infants whose mouths have been subjected to cleansing procedures; those who have not been so maltreated do not have them. Evidently, therefore, they develop purely from mechanical injuries of the mucous membrane due to cleansing. The ulcers are shallow excavations, gray to yellow in color, and cause no complaint whatever, nor do they interfere with nutrition.

Treatment.—Mechanical cleansing of the mouths of nurslings is not good practice. The ulcers will heal without any treatment and without cicatrization.

Lingual Ulcers.—Infants frequently suffer from ulcers of the lingual frenum which are due to erosions caused by the sharp edges of the lower middle incisors while the infant is nursing or coughing. They will not occur when the edges of the teeth are smooth. In infantile pertussis these ulcers are of very frequent occurrence.

Treatment.—Sharp-edged teeth must be filed down or removed.

Traumatic Tumors.—Traumatic tumors of the frenum sometimes occur in nurslings. They are clearly the result of chronic injury to the frenum through the incisors or the hard edges of the gums. Microscopically they are seen to consist of connective tissue covered with a strongly developed epithelial layer. The growths are benign and give rise to no symptoms.

Treatment.—Extirpation of the tumor and removal of the irritating teeth.

STOMATITIS.

Simple or Catarrhal Stomatitis.—The mouth in healthy individuals with sound teeth may remain free from pathologic manifestations even without any particular care being bestowed upon it. Stomatitis simplex is very often due to carious teeth and excessive calcareous deposits. The parts of the gums adjoining these deposits become easily inflamed and swollen, presenting a red or blue-red discoloration. Bulging and loosening of the margins of the gums may increase, accompanied by exaggerated salivation, and the teeth may drop out. This condition is often associated with excessive *fetor ex ore*. It may also be followed by a complete involvement of the oral mucosa, coating of the tongue, and interference with mastication and speaking. There may even be fever. Individuals suffering from nephritis, diabetes and other constitutional diseases may develop stomatitis even though their teeth are sound.

Treatment.—Unsound teeth are to be removed, and the oral cavity and the teeth carefully cleansed by mechanical means. Antiseptic mouth-washes, such as thymol (1:2000), acetate of aluminum (1:200-500), or permanganate of potassium (1:2000-5000), usually effect a cure in a short time. Inflamed points, ulcers and the margins of the gums may be rubbed with iodoform mass (see page 298). The constitutional affection is to be treated according to general indications.

Gangrenous Stomatitis. Inflammatory processes in the mouth easily lead to necrosis and ulceration. In a large number of cases of severe ulcerating stomatitis, the ulcers and the secretion have been found to contain the same bacteria as those which are considered responsible for Vincent's angina. It is justifiable, on the ground of these very frequent findings, to regard these micro-organisms as the cause of ulcerating stomatitis.

The most frequent form of gangrenous stomatitis is the ulcerating.

Stomatitis ulcerosa occurs very frequently in children between the ages of five and ten; it may occur at any other age, but only when teeth are present. It is found in cases of neglected dental hygiene, carious teeth, and excessive accumulation of calcareous deposits, and may easily be the consequence of grave disorders of nutrition (diabetes, scrofulosis, rachitis, scorbutus). Mercurial stomatitis, which has been described above, likewise belongs to *stomatitis ulcerosa* according to its clinical course. The affection commences with the gums, which become swollen, spongy and bleeding. As a consequence, the margins of the gums and the malar mucosa opposite develop irregular, sharply demarcated, whitish yellow patches, beneath which a purulent fluid may accumulate. This constitutes an epithelial necrosis, leading to the formation of ulcers with a yellowish slimy base and surrounded by serrated, bulging, bluish-red margins. The ulcers may spread to the gums and the floor of the mouth, penetrating rather deeply into the tissue. This condition is accompanied by a peculiar putrescent odor. As the affection proceeds, the distress will increase, notably in masticating, swallowing, and speaking. The tongue, cheeks, and possibly the soft parts of the neck may be highly edematous. Under certain circumstances general sepsis follows as infiltration of the soft parts of the buccal fundus and the neck proceeds.

Treatment. Removal of the cause, so far as possible (carious teeth), and omission of mercury. Efforts should be made to remove the concretions and the decomposed and necrotic masses. This will be best accomplished, especially in the presence of considerable pain, by rinsing the mouth with tepid antiseptic fluids by means of an irrigator. Astringent and antiseptic mouth-washes are at once indicated if the patient is able to attend to their application himself. Such remedies are: Borax (1 : 30), permanganate of potassium (1:2000-5000), tincture of myrrh, and tincture of krameria. Hydrogen peroxid is especially useful, being very efficacious in loosening the necrotic masses. In progressive ulceration, iodoform is the best antiseptic, the diseased parts being rubbed with iodoform mass, and iodoform gauze inserted in appropriate places (see page 298). Based upon the favorable results obtained in a few cases of Vincent's angina with arsphenamine, this remedy should also be borne in mind in the treatment of ulcerous stomatitis.

Noma.—The gravest form of gangrenous stomatitis is noma, a rather rare affection. A characteristic inciting factor has not yet been discovered, although spirochete and fusiform bacilli have been found, as in ulcerous stomatitis. Noma is preëminently a children's disease and is often preceded by measles. It has also been observed after scarlet and typhoid fevers, pneumonia, and after the administration of mercury. Usually the affected children are in a poorly nourished condition. The affection commences

with salivation, *fetor ex ore*, and the signs of simple stomatitis. Then follows in most cases the formation of a vesicle, at first bluish-red but later blackish, at a point between the angle of the mouth and the orifice of Stenon's duct opposite the first and second molars. There is an objectionable odor. The cheek becomes swollen and pale. The swelling increases, extending upward toward the eye and nose. Finally the infiltration is as hard as a board. Simultaneously the necrosis extends over the mucous membrane. The black, discolored portion develops into a brownish-black area, rapidly involving the entire surface of the malar mucosa. Outward perforation threatens, as evidenced by a bluish-red spot, increasing in extent, which finally becomes gangrenous and perforates. The perforation gains in extent, due to tissue disintegration, and may involve the entire area from the angle of the mouth over the upper and lower maxillæ to the ear, finally exposing the bone behind the ear. The gangrenous process may now limit itself, in which case the necrotic mass is gradually desquamated, and the defect is gradually covered with granulation tissue, leaving in the end an exceedingly disfiguring scar. In about 70 per cent. of the cases death results from inanition or sepsis. The duration of the disease is usually from eight to fourteen days, and its prognosis is always very grave.

Treatment.—In the initial stage an attempt should be made to extirpate the pathologic focus, but in spite of extensive excision the gangrene often persists in its course. After extirpation, Paquet's scarification may be resorted to; this failing, the treatment must be confined to the relief of pain, regulation of the diet, and removal of the ichor and gangrenous masses by irrigation. Patients must be isolated, owing to the pestiferous odor they exhale.

Erysipelatous Stomatitis. It is only in rare cases that erysipelas spreads from the facial skin to the oral cavity. When this does occur, there is diffuse, intense reddening, desiccation and painfulness of the mucous membrane. The uvula may be swollen. The pain may render swallowing impossible. Stomatitis erysipelatosæ can be assumed to exist with certainty when erysipelas has preceded the attack or follows it. The tongue may swell considerably.

Treatment.—Careful oral hygiene, small pieces of ice, scarification of the mucous membrane, and if necessary artificial feeding. Colloidal electro-silver is administered intravenously with good results.

Aphthæ.—This affection is characterized by the early appearance, with febrile manifestations, of pale yellow patches, either singly or in large numbers. They may be very small or as large as a small pea, and surrounded by a slightly elevated, very red fringe. They are sharply demarcated, round or oval in shape. In most cases they undergo rapid resolution by becoming detached from their base, the epithelium simultaneously growing over the traumatic

surface from the surrounding area. The affection usually takes a tedious course, numerous new foci making their appearance. It attacks all parts of the mouth, least often the gums. Each individual focus is intensely painful, often interfering with mastication, deglutition, and articulation. Coincidentally the patient suffers from a moderate degree of general stomatitis, exaggerated salivation, fever, and more or less malaise. Aphthous stomatitis might also be designated stomatitis maculofibrinosa; its cause has not yet been discovered with certainty. Staphylococci are not infrequently present. In most cases the affection is confined to infants from ten to thirty months old, although nurslings and adults are also subject to it. In spite of the serious discomforts which attend the disease, and its frequent wide extent, it usually heals spontaneously; but it may be contracted many times.

Treatment.—Considering that the affection undergoes spontaneous healing, no treatment is required except painting with weak antiseptic solutions (boric acid, permanganate of potassium, boroglycerid). These remedies are applied to the mucous membrane without the use of force. When the pains are severe, anesthesin, orthoform or apothesine powder may be insufflated (see page 270).

Chronic Aphthæ (Aphthæ Tropiciæ).—Chronic recurring aphthæ are very rare. They were first described by Mikulicz. Small epithelial defects, the size of a millet seed, or small vesicles, appear at the lingual margin at intervals of four to six weeks. Within four or five days they attain to the size of a small pea. The number of these defects varies considerably; sometimes there is only one, sometimes two or three, rarely more. Their appearance is associated with slight general stomatitis and salivation. The affection heals spontaneously without cicatrization; but it frequently recurs, even before the old foci have quite healed. The aphthæ are very sensitive to the touch. A very similar affection occurs in patients suffering from "sprue," or chronic tropical diarrhea.

Sprue.—Sprue or psilosis is a disease of tropical and subtropical countries which renders the mucous membrane of the mouth and tongue very sensitive and superficially ulcerated. That it is of infectious origin is shown by its occurrence in people closely associated with one another. Examination of the stools shows a decrease or absence of the digestive ferments; the stools are diarrheic, of a soft, pasty and fermentative character. It is believed that the thrush fungus (*Monilia Albica*) may be the cause of the condition. The disease usually runs a chronic course. Recent reports show that sprue has been treated successfully with emetine hydrochlorid (see page 723). The striking feature in sprue is the persistence of the complete absence of pancreatic secretion. The pancreatic achylia implies that the administration of pancreatic extract by mouth is essential in the treatment of the disease.

Thrush.—This affection of newborn infants is caused by the thrush fungus, a widely disseminated form of vegetation which is found in milk as well as in the mouths of healthy nurslings. In the mouth it occurs as a round or oval yeast-like structure with ramifying threads. Healthy adults and nurslings are not affected by this fungus; though it may be present, they are immune. In the newborn the slightest disorders of nutrition are sufficient to allow of the pathogenic development of the thrush organism. In adults the disease rarely develops except after protracted fevers, such as typhoid. Thrush is at first manifested by the appearance of small white specks on the mucous membrane of the cheeks, gums, lips, and the dorsal surface of the tongue, which soon grow and confluence into membranous deposits, adhering rather firmly to the mucous membrane. In order to establish the diagnosis, it is necessary to wipe off a portion of the deposit and examine it under the microscope in concentrated solution of potash. If the constitutional disease is not too severe, thrush remains confined to the mouth, but in grave cases and in unconscious patients it may spread to the pharynx and esophagus. In most cases the growth is confined to areas without cylindric epithelium. It is apt to penetrate rather deeply into the mucous membrane.

Treatment. The treatment is directed to the underlying disease; the thrush will disappear as soon as this is removed. It is not advisable to remove the coating mechanically, since it is situated too deeply in the epithelium to admit of eradication in this manner. Weak antiseptic solutions may be used. For infants, boric acid powder may be wrapped up in a small bag, which, after having been dipped into sugar or saccharin, is given them to suck.

SYPHILIS OF THE MOUTH.

Primary Syphilis.—The primary affection is usually located on the lower lip, at the tip of the tongue, or at the tonsils, but it has been observed at other points of the oral mucosa. The presence of white triangular patches on the labial mucous membrane is often indicative of syphilis. It may be assumed that the infection does not occur unless the syphilitic virus is conveyed to lesions of the mucous membrane. This may happen in various ways—for instance by kissing, by the use of infected instruments (glass-blowers) or eating and drinking utensils, or by unnatural coitus. The primary manifestation in syphilis of the mouth (chancre) develops in the same way as on the genitals. There is a small superficial epithelial defect, with a rampart-like thickening of the margins and cartilaginous hardness at the base. In most cases there is only one sclerosed spot; multiple sores are the exception. Should secondary infection occur, the primary affection of the tonsils may lead to deep ulcers with extensive disintegration. Of diagnostic impor-

tance is the swelling of the regional lymph glands. In contrast with the small sclerosis, these are often considerably swollen, indurated, but painless. The complaints occasioned by the primary affection are usually slight, but the considerable tension of the soft parts caused by the sclerosis is unpleasant. Swallowing and speaking are difficult. As a rule the diagnosis is not difficult except when the tonsils are the site of the infection and induration is absent. In doubtful cases the diagnosis is made with the aid of the Wassermann reaction, by exploratory excision (when carcinoma is suspected), and *ex juvantibus* from the effect of arsphenamine causing rapid resolution in as short a time as two or three days. The diagnosis also follows from the demonstration of the *Treponema pallidum* in the secretion of the scraped ulcerous surface. The primary affection heals without leaving any material scars.

Treatment.—Antisyphilitic treatment should not be delayed after the diagnosis has been established. It has been generally believed to be a matter of indifference as regards the course of the syphilitic infection whether treatment was instituted upon the appearance of secondary manifestations or at an earlier stage. More recent experience, however, points to the probability that very early arsphenamine treatment (see page 534) in conjunction with mercury is particularly efficacious and likely to positively arrest the course of the syphilitic affection. From a prophylactic point of view the attention of the patients should be directed to the importance of preventing infection by kissing or by the common use of eating and drinking utensils. For local treatment the application of calomel or iodoform is advisable. The swollen lymph glands undergo rapid resolution when rubbed with mercury ointment or covered with mercury plaster. Typical Hunterian chancres are not so common as was formerly believed. The dark field method of examination has shown the typical treponemata in various atypical sores.

Secondary Syphilis.—The secondary manifestations occurring in the mouth correspond exactly to those of the skin, but present peculiar pictures owing to the soil upon which they develop. The first manifestations of secondary oral syphilis often include an erythema occurring in small or large patches upon the oral mucosa, especially on the palate (angina erythematosa luetica). This erythema usually disappears in a few days. The most frequent secondary manifestations are the mucous plaques (*plaques apalines*), consisting of circumscribed infiltrations of the submucosa, of roundish shape, which are sharply demarcated and raised flat above the mucous membrane. The epithelium above becomes gradually muddy, giving rise to the typical patches. These consist of a grayish-white thickening on the slightly swollen mucosa, with a hyperemic marginal zone, the upper surface of which has a characteristic pearl-white to milk-white velvety appearance. When the plaques have persisted for a long time they become

opaque. As soon as the muddy epithelium becomes detached, flat defects of the mucous membrane with a red or yellowish base remain behind. The plaques are usually situated on the mucous membrane of the lips, cheeks, and tongue, and, when long persisting, assume variable roundish, oval or other shapes and sizes. Their greatest extent is reached at the palatal arches. Occasionally the plaques will spread from the lips to the adjacent skin, where they form papules covered with crusts. Carious teeth, the pressure of artificial dental plates, highly spiced food, alcoholic beverages, and excessive smoking, favor the development of the plaques and their recrudescence. The complaints occasioned by the plaques are often very slight and again very troublesome, but in any case the plaques are most dangerous sources of infection for communicating syphilis to other persons. For this reason the diagnosis and corresponding instructions to the patient are of the utmost importance. The diagnosis of characteristic plaques is easy, and is supported by the demonstration of syphilitic skin eruptions, glandular swelling, the findings of the Wassermann test, and, if the disease be present, by the effect of antisyphilitic medication.

Treatment.—In the first place, constitutional antisyphilitic treatment is required—mercury for inunction or injection, and arsphenamine, which apparently has an exceedingly favorable and rapid effect upon these syphilitic affections of the mucous membrane (see page 534). For local treatment it is advisable to touch the plaques with a 1-per-cent. sublimate solution, 10- to 20-per-cent. silver nitrate solution, 5- to 10-per-cent. chromic acid solution, or the silver nitrate stick. When the plaques are very painful they may be powdered with anesthesin. The above solutions are best applied with wooden sticks tipped with cotton, which are to be thrown away after use. The argentum stick is contra-indicated in non-syphilitics. Scrupulous oral hygiene, regular use of mouth-washes and gargles, and elimination of all irritations, form part of the treatment (see page 296).

Tertiary Syphilis. The tertiary processes in the mouth occur at the hard and the soft palate, at the margins and dorsum of the tongue, less often at the malar mucosa and lips. When the lips are affected they exhibit circumscribed gummata which may disintegrate and cause diffuse inflammatory infiltration, with considerable and firm swelling of the lips. Palati gummata, which are of very frequent occurrence, may spread from the hard palate to the periosteum, rapidly leading to necrosis of the thin osseous plate. The gumma of the hard palate commences as a small nodule which gradually becomes larger and softer, presenting a bluish discoloration. It soon disintegrates, the mucous membrane is perforated, and ulcers of different sizes are formed with sharp, coarse margins and a yellow lardaceous base, exposing the raw necrotic bone beneath. The sequestre are separated, per-

forating the hard palate. As a result there will be a defect of variable size left in the hard palate after healing has taken place. The extent of the destruction may be slight or so great that swallowing and speaking are considerably interfered with. Gummata of the soft palate develop in a similar way; they are often multiple and may also be located on the palatal arches and the uvula. The tongue is less often involved, and in men (on account of the use of alcohol and tobacco) the lingual involvement usually takes the form of sclerotic glossitis, with very firm infiltration and a perfectly smooth mucous surface, owing to complete loss of the papillae. This form of glossitis is complicated by deep rhagades and erosions, usually at the lingual margins, which are apt to give rise to marked subjective symptoms. After healing, they leave calluses at the surface or in the parenchyma. The deeply situated calluses may cause deep furrows in the tongue. Lingual gummata may likewise occur, leading to considerable enlargement of the tongue and extensive ulceration. The tertiary manifestations of the tongue may heal spontaneously, but are subject to frequent recrudescences.

Treatment.—The specific constitutional treatment is fully described in Chapter XXVIII. In deep ulcerations and bone necrosis, local treatment is necessary in the form of iodoform mass and tamponade with iodoform gauze (p. 298). In glossitis luetica it is necessary to repair carious teeth and to remove calcareous deposits. Rhagades are painted with silver nitrate or chromic acid solution. The mouth must be rinsed often with antiseptic washes. Alcohol and tobacco are forbidden. The palatal defects can be repaired by plastic operation, as long as they are small; larger ones have to be closed by plates.

In some cases the salivary glands of the buccal fundus and those at the tip of the tongue show tertiary syphilitic symptoms in the form of swelling and enlargement.

TUBERCULOSIS OF THE MOUTH.

As a rule the susceptibility of the oral mucosa to tuberculous infection is not great. Primary oral tuberculosis is very rare, but may be caused by infected food (milk), infected eating and drinking utensils, or infected fingers. The infection may result from the bovine as well as from the human type of tubercle bacilli. Latterly the fact has been emphasized that the bovine type plays a far greater rôle in the infection of man than was formerly assumed. The mucous membrane of the mouth may acquire tuberculosis secondarily by direct extension from the facial skin or by tuberculous sputum.

Lupus of the Oral Mucosa.—Lupus presents the same characteristic features on the mucous membrane of the mouth as on the

external skin. Points of predilection are the free margins of the lips, the vestibulum oris at the superior maxilla, the palatal vault, and the soft palate. There are at first characteristic nodules, which later increase considerably in number, growing in close proximity. When the epithelium has been lost, there appear flat ulcerations which may merge, the secretions drying up into crusts. Their margins are lardaceous, often undermined, and permit the recognition of small nodules. The disease persists for a number of years. Isolated parts may heal while the pathologic process continues at the margins. Extensive destruction of the oral structures may take place in the course of time from this cause.

In tuberculosis of the tongue there occur infiltrations, rhagades, and ulcerations. This affection has a comparatively favorable prognosis. In grave cases of tuberculosis all parts of the mouth are subject to ulcerations and miliary tuberculous nodules, with simultaneous infiltration of the deeper tissue. Extensive changes of this kind are found on the tongue and lips, notably also in the pharynx and on the palate and tonsils. The prognosis generally is very unfavorable. The discomforts may be slight, or, on the other hand, very tormenting, interfering with speech and nutrition. In making the diagnosis, this affection may be mistaken for syphilis or carcinoma, and in order to clear up the point it is important to demonstrate tuberculous changes in other organs or tubercle bacilli in the pus, or apply the von Pirquet test.

Treatment. In the absence of pronounced tuberculosis in other organs, notably the lungs, energetic local treatment is indicated. Whatever infiltrations or ulcers lend themselves to extirpation, according to position, seat and size, should be removed with the knife or the Paquelin cautery. The after-treatment consists in applying iodoform to the wound. Large ulcers and necrotic parts may be tamponed with iodoform gauze. Great pain may be relieved by the local application of orthoform or anesthesin or the administration of morphia. Latterly, lupus of the mucous membrane of the mouth has been treated with the Roentgen ray by means of special tubes adapted to the purpose. This procedure demands great caution in order to prevent roentgenic ulcers, which heal with great difficulty. The Finsen light does not act so well on lupus of the mucosa as on that of the skin. Application of sunlight, and treatment with hot air, steam, and carbon dioxide snow, are new methods which deserve to be tested.

GLANDERS OF THE MOUTH.

Malleus.—Glanders in man is a very rare affection. The majority of patients are employed in handling horses. The disease may run either an acute or a chronic course. The acute form results from direct infection of the blood current through open wounds,

causing infiltrates which usually extend over large areas of the oral mucosa. The chronic form results from extension of the infection through the lymph current, starting from a primary glanderous focus on the skin in the neighborhood of the nose or mouth. The ulcerations frequently attack the entire hard palate, sometimes involving the soft palate also. In rare cases the affection extends to the deep parts of the upper lip, destroying large portions, both of the lip and of the nose. Infiltrations also occur in the lower lip and are often accompanied by infiltrations of the lymph glands of the neck. The diagnosis is always difficult and cannot in all cases be made by mere inspection of the pathologic foci. It is important to observe that, almost without exception, glanders of the mouth is associated with a similar eruption on the skin, which, however, may have already healed. Actively proliferating granulations are always found in the anterior part of the nose. The glottis is often involved. Histologic examination of the ulcers shows simple granulation tissue without caseation of the glandular cells—an important fact in differentiating the disease from tuberculosis. Absolute proof of glanders is, of course, the demonstration of the glanders bacilli, which are best obtained from scraped superficial parts of the ulcers and granulations, and are demonstrated by intraperitoneal inoculation of guinea-pigs, the latter showing typical glanderous periorchitis in four or five days. One reason why a reliable diagnosis is of great importance is that these patients constitute a grave danger to others. The complaints are usually slight, even in advanced cases. There is anorexia and lassitude; nutrition is little interfered with, and there is no fever. There are often septic complications or glanders pneumonia. The prognosis is bad. The great majority of cases terminate fatally, the acute ones always.

Treatment.—The ulcers of the mouth are treated locally in accordance with general rules. The constitutional condition is said to be sometimes favorably influenced by mercury and potassium iodid. Serum therapy has not yet been successful, nor has mallein, prepared on the principle of Koch's tuberculin. Vaccines of killed glanders bacilli seem to exert a favorable influence upon the constitutional affection in some cases.

LEPROSY OF THE MOUTH.

In nearly all forms of leprosy the mucous membrane of the mouth becomes involved—in the tuberculous form, for instance, in about 70 per cent., according to statistics. The involvement occurs at an early stage, but, owing to the slight subjective symptoms, as compared with the grave skin affection, is often overlooked. The bacteria of leprosy find a very favorable culture ground in the mouth, where the presence of lepra bacilli can be

demonstrated in larger numbers than anywhere else. For this reason, leprosy of the mouth gains in importance by being an easy source of infection to others. The diagnosis is usually not difficult, because in most cases the skin is simultaneously involved. The lepra bacilli can be demonstrated by means of the ordinary tubercle stain. The symptoms occasioned by the oral affection are strikingly slight, large infiltrations sometimes causing very little inconvenience. This is, as a rule, due to the fact that anesthesia of the affected parts occurs at an early stage. Salivation is often excessive. The prognosis is bad. A spontaneous cure is a rarity. Nevertheless, leprosy infiltrations may persist for years without increase.

Treatment. Ulcerations often heal readily under local treatment (nitrate of silver, chromic acid); internally, sodium salicylate and chaulmoogra oil. The latter, diluted with olive oil in equal parts, may also be injected intramuscularly. Sodium gynocardate, made from the gynocardic acid of chaulmoogra oil, is administered intravenously with reported good effect. The leprosy serum of Carrasquilla, lupolin as recommended by Rost, and the nastin of Deycke (prepared from *Streptothrix leproides*) have proved failures.

SCLEROMA

Scleroma, a hardened patch or induration, prevails in Russia and Galicia, and is found sporadically throughout the world. It seems to be gradually spreading. Von Frisch's capsule bacilli have been regarded as the cause of scleroma, but more recently the capsule bacteria have been found to be identical with Frieslander's pneumonia bacilli. They grow readily upon agar.

The affection is very chronic; it may last for decades, the subjective symptoms being comparatively slight. The diagnosis may be made from observing the course of the disease or from demonstration of the capsule bacteria. The prognosis is very bad. Death may result even after many years by continuous advance of the process and extensive cicatrization which may occlude the entrance to the esophagus and glottis.

Treatment.—Radical extirpation of the diseased parts may be attempted, medicinal treatment being a failure. The use of the Roentgen ray has been attended with favorable results in scleroma of both nose and mouth, causing complete resolution in some cases. The treatment has to be continued, however, for months or years. Radium likewise seems to have a favorable effect. Application of the Finzen light has not been successful. Benefit has been observed to follow accidental complication with erysipelas.

ACTINOMYCOSIS.

There is no doubt that the mouth is the most frequent port of entrance for actinomycosis in man, and the disease is therefore most frequently found in the mouth, throat, and glottis. The most reasonable explanation is that the fungus is conveyed to the mouth by plants, possibly from using straws or grass stems (from pastures where there are diseased cattle) as tooth-picks. In some cases the infection is transmitted directly from diseased cattle.

In the majority of cases the alveoli are attacked first, or there may be a periostitic abscess. The fungus has also been demonstrated in carious teeth. Thus in man there is a periostitis alveolaris, while in animals there is a central affection of the inferior maxilla. Though the primary periostitis heals, the disease continues as an actinomycotic tumor or a submaxillary abscess, usually situated between the inferior maxilla and the hyoid bone. Metastases are therefore often found between the muscles and connective tissue of the neck. The tonsils, palatal arches and salivary glands are not often involved in man. The tongue, however, is often affected. There are hard, sharply circumscribed infiltrates, mostly at the tip of the tongue, which rarely ulcerate. These infiltrates are often difficult of diagnostic interpretation, and it may become necessary to resort to an exploratory incision or excision to demonstrate the presence of the fungus. There also seems to be a possibility of abscess formation in the tongue, causing violent pain and dyspnea.

Treatment. The pathologic foci are exposed as freely as possible. There is no need, however, for extensive radical surgical measures, because the prognosis of actinomycosis is by no means bad, and occasionally the affection even heals spontaneously. The object of exposure is to disturb the fungus, which is anaërobic in its ordinary way of living. The wounds are kept open by tampons of iodoform gauze. In acute suppurative infiltration, simple incision is sufficient. Lingual tumors are best extirpated. When the course is chronic and the infiltration considerable, surgical intervention alone will not suffice. Good results have been obtained with potassium iodid; 1 to 3 Gm. (15 to 45 grains) daily is administered internally, and a 5-per-cent. solution is injected into the foci. It is advisable to combine potassium iodid with the surgical treatment.

SKIN DISEASES IN THE MOUTH.

Affections of the mouth analogous to those of the skin are caused either by direct extension from the latter, or as isolated affections previous to the appearance of the skin disease, or even without the latter appearing at all.

Eczema.—Eczema very often occurs on the lips. Scrofulous individuals or those with a tendency to the formation of exudates of various kinds, notably children, and persons harboring latent or manifest tuberculosis, are predisposed to this affection. Patients of this class very often suffer from chronic rhinitis. The acrid nasal secretion easily leads to eczema around the nose, which spreads directly to the labial mucosa. Ulcers around the angles of the mouth in children should also be classed as eczema. They are exudative plaques and are covered with crusts, owing to constant scratching and licking. Healing requires several weeks, and there is great tendency to recrudescence.

Treatment.—The treatment is directed to the underlying cause, if any, and to the rhinitis. The affected parts of the skin and mucous membrane, especially rhagades, are painted with a 5- to 10-per-cent. solution of silver nitrate or with mercurial salve (white precipitate, 1 per cent.). Frequent washing with soap should be avoided; mouth-washes containing volatile oils are rigorously contra-indicated. Arsenic is indicated for internal administration. A correction of diet, as shown by the anaphylactic food tests, is productive of good results.

Lichen Planus. Lichen ruber planus occurs in characteristic form in the mouth. In about 50 per cent. of all skin cases the mouth becomes involved. There are the well-known lichen nodules of the lips, malar mucosa, tongue, hard and soft palate. Their size ranges from a pinhead to a pea; they are whitish, very coarse, and grow in isolated groups forming circles or curves. Epithelial defects or considerable inflammatory manifestations are absent. The affection causes practically no subjective symptoms whatever.

Treatment.—Lichen of the oral cavity is favorably influenced by the internal or subcutaneous use of arsenic, but more slowly than the skin affection. There is no need for local treatment.

Lupus Erythematosus.—Lupus erythematosus is located on the nose and both cheeks, and can easily be communicated to the oral cavity, and notably the lips. This results in gray-white reddish plaques, surrounded by a narrow blue-red fringe. Sometimes there are erosions partly covered with crusts, and loss of epithelium. The diagnosis in the presence of the characteristic affection of the skin is easy. The course of lupus erythematosus of the mucosa is just as chronic as that of the skin.

Treatment.—The affection is not readily influenced by medication, but there is some prospect of benefit from energetically treating the surface with the Paquelin cautery. When the lips are involved, the application of a highly concentrated preparation of ichthyol is advisable.

Pemphigus.—Pemphigus of the skin very often leads to involvement of the mucous membrane of the mouth, causing circumscribed epithelial "muddy" areas and white-gray deposits resembling

diphtheritic membranes. The deposits become detached after having persisted for a long time, leaving sharply demarcated patches behind. These may fuse and finally cover large areas, or lead to erosions and shallow ulcers. Pemphigus of the mucous membrane, therefore, is not so characteristic as that of the skin, for only exceptionally do real vesicles form in the mouth, owing to its moisture. An isolated pemphigus of the oral mucosa without a corresponding affection of the skin is very rare and, when it occurs, very obstinate. When extensive it causes severe pain, dysphagia, and *fetor ex ore*; the entire mucosa may become involved under certain circumstances. The participation of the oral cavity in pemphigus of the external skin is a very unfavorable sign. The diagnosis is not difficult when cutaneous pemphigus is present.

Treatment.—Healing rarely takes place, and treatment has but little effect. Isolated areas may heal for a time, assisted perhaps by painting with 20-per-cent. silver nitrate or tincture of iodine. Aside from these measures, the treatment is confined to the relief of pain by painting with a local anesthetic solution (cocain, eucain, or novocain) or by powdering with anesthesin, orthoform or apothesine (see page 270).

Erythema Exudativum Multiforme. In comparison with the characteristic cutaneous manifestations, those of the mucous membrane are less distinct. There are roundish remnants of vesicles in the shape of detached epithelial shreds or easily bleeding yellowish deposits. The affection is benign and does not require any particular treatment.

Herpes.—Herpes zoster may occur at any point in the mouth, especially the cheeks, pharynx, or tongue, as well as in the gums. It is always unilateral, and manifests itself in vesicles grouped on the reddened and swollen mucosa. The covering of the vesicles is easily destroyed. The affection is associated with violent neuralgic pains, and recrudescences are not infrequent. It may occur in the mouth without any simultaneous affection of the skin. The diagnosis can easily be made in most cases from the acute beginning, painfulness, unilateral occurrence, and the appearance of the vesicles as described. The treatment is hygienic (see page 296).

Urticaria. Urticarial eruption in the mouth is very rare. It occurs in the form of circumscribed, lustrous, usually persistent, recurrent swellings, without any pronounced inflammation. Angioneurotic edema may likewise occur in the mouth, at the base of the tongue.

Scleroderma.—The involvement of the mouth is comparatively rare and is often overlooked. There are doughy swellings and indurations of the mucous membrane, notably of the tongue. High degrees of the affection lead to ulceration.

LEUKOPLAKIA.

This is an affection of the mouth which is characterized by a focal thickening of the epithelium of the mucosa in circumscribed areas, due to abnormal cornification. The affected parts form a kind of ridge resting upon a mucous membrane that is abundantly supplied with vessels, strongly infiltrated with leukocytes, and the papillae of which are narrower, longer and more numerous than normal. These ridges appear as smooth, dry, milk-white patches. The most recent look like mucous membrane which has been painted with weak silver nitrate solution. The older the foci, the stronger and firmer the ridges, which finally become a pure white or bluish-white, with a kind of mother-of-pearl luster. The plaques are sharply separated from the neighboring parts, and often surrounded by a narrow infiltrated zone. Plaques in the various stages of development may be present in the mouth at the same time. Here and there a ridge is raised at the margins, and finally detached, giving rise to rhagades or deep tears. Leukoplakia preferably invades the anterior part of the dorsal surface of the tongue, especially toward the tip and at the margins. There are also foci on the inner malar surface and the lips. Foci of the palate and alveolar process are very rare.

The disease runs a very chronic course, several decades being sometimes required for the originally tender epithelial opacities to develop into thick ridges. It is almost entirely confined to the male sex and rarely appears before the fortieth year—as a rule not before the fiftieth or sixtieth. We know now that it is an independent affection—an important discovery in view of the fact that formerly it was regarded as syphilitic and treated accordingly. It is positively not syphilitic, although there is no doubt that the presence of syphilis predisposes to its development. Etiologically, excessive smoking and the free use of strong alcoholic beverages and highly spiced food are of importance; hence the relatively rare occurrence of the disease in women. Gastric and intestinal diseases likewise predispose to it.

Symptoms.—The complaints are not excessive. Pain does not occur except in extensive rhagades; otherwise patients experience a peculiar blunted sensation, as if they held a foreign body in the mouth. For this reason the tongue is constantly licking and palpating, thus mechanically tearing off the ridges. These patients are prone to become hypochondriac, either because they consider the affection syphilitic or because they fear the development of carcinoma. As a matter of fact, carcinoma does occasionally develop on the site of leukoplakia; this observation has been frequently made. But the sequence is, fortunately, rather rare.

Treatment.—The exciting cause has to be eliminated; excessive smoking, strong alcoholic beverages and highly spiced dishes

must be avoided. Defective teeth must be repaired. When the inhibition of nicotine cannot be enforced, cigars or cigarettes should be smoked through holders. Chewing tobacco is, of course, to be forbidden.

Light cases are treated with mild mouth-washes, if for no other reason but to pacify the patient. Various mouth-washes had best be used alternately. Should patients complain of troublesome burning sensations, the mouth may be rinsed with a decoction of althea root or of Iceland moss. The plaques may be removed by caustic agents: silver nitrate either pure or in a 50-per-cent. solution, 5- to 10-per-cent. lactic acid, or 30-per-cent. peroxid of hydrogen. Salicylic acid acts as a solvent upon the horny layer; also resorcinol in 2-per-cent. solution. Pure balsam of Peru, applied with a brush and kept in the mouth for two or three minutes, has a good effect. The alkaline mineral waters have been recommended. This medication, however, means failure in most cases, with resulting psychic depression on the part of the patient, especially when the oral condition is painful and there is trouble in eating, drinking, and speaking. In these cases surgical measures may be attempted as a final resort. Radium has proved of great value.

Many authors suggest antisyphilitic treatment, and in some cases energetic mercurial medication has resulted in complete cure. Latterly, arsphenamine has been warmly recommended. Antisyphilitic treatment is certainly indicated unless there are reasons to the contrary.

ANIMAL PARASITES IN THE MOUTH.

Larvæ of flies, cysticerci and echinococci have been found in the mouth, and trichinæ in the tongue.

NERVOUS AFFECTIONS OF THE MOUTH.

Paralysis of the Facial Nerve.—Paralysis of the facial nerve often induces an oblique position of the palate; the troublesome flow of saliva is due to the paralyzed angle of the mouth being constantly open. Injuries to the hypoglossal nerve lead to paralysis of the lingual musculature. There are to be distinguished: Total glossoplegia, glossoplegia interna (longitudinal and transverse lingual muscles), and glossoplegia externa (geniohyo-, stylo-, hyo-, palato- and chondro-glossus muscles). Some lingual muscles are, occasionally, paralyzed by neuritis, as after diphtheria. Convulsions of the hyoid musculature may easily occur, especially in epileptics, causing the tongue to be bitten. Isolated clonic and tonic spasms are observed in hysteria. Tonic spasms are also met with in tetanus, rabies, hemiplegia, and hemiparalysis agitans.

Clonic spasms occur in cortical affections of the brain, chorea, myotonia congenita, and paramyoclonus multiplex. Occupation spasms of the tongue have also been observed. Ataxia of the tongue occurs in tabes. Permanent contractures of the genioglossus and styloglossus muscles occur in hysteria.

Disorders of sensation are usually found together with similar disorders in other trigeminal regions. Anesthesia is usually confined to one half of the tongue and is often not discovered by the patient. Disorders of the sense of taste due to lesions of the chorda tympani are confined to the anterior part of the lingual margin. Affections of the glossopharyngeal nerve impair the sense of taste in the posterior part of the tongue.

By neuralgia of the tongue is distinctly understood neuralgia of the lingual nerve which represents a special form of the genuine trigeminal neuralgia. This is accompanied by typical paroxysms of pain, both spontaneously and upon extraneous irritation. The neuralgia is rarely confined to the lingual nerve; as a rule there is also neuralgia of the inferior maxillary. Neuralgic pain in the region of the lingual nerve should always suggest local affections of the tongue, especially carcinoma.

Treatment.—The treatment corresponds to that of trigeminal neuralgia. Locally, the galvanic current may be tried, or, if necessary, resection of the lingual nerve.

Glossodynia. Glossodynia comprises indefinable painful sensations in the tongue which may torment the patient for hours or days. It is a condition, therefore, which differs absolutely from neuralgia. Patients complain of all kinds of painful sensations in the tongue, for which they call in the physician. In most cases they are hypochondriacs who are apprehensive of lingual carcinoma and syphilis. Deglutition is not impaired. They are able to eat well, and during eating forget their complaints.

Treatment.—The treatment should be psychic. No local treatment is indicated.

VASOMOTOR, TROPHIC AND SECRETORY DISORDERS.

In tabes and syringomyelia there are ulcers of the alveolar process and hard palate (*mal perforant buccal*). In Raynaud's disease, angiospasm occurs in the tongue by way of exception. Reflex salivation occurs in hysteric and neurasthenic patients following painful affections of the mouth. Decreased salivation occurs in acute febrile affections, likewise in hysteria and paralysis of the sympathetic. Total arrest of salivation, called *xerostomia*, is a tormenting, painful condition, causing difficulty in swallowing and speaking. Absolute toothlessness seems capable of causing it. In some cases pilocarpin has proved useful.

AFFECTIONS OF THE TONGUE.

Malformation.—It occasionally happens that the thyroglossal duct remains patent, causing the base of the tongue to swell from congested secretion. This condition requires surgical intervention. The formation of cysts in the duct is also possible. Adhesion of the tongue to the palatal fundus likewise occurs. A short frenum lingue is no malformation, and the incision of the same which is frequently practiced is superfluous and objectionable. Disorders of speech are never improved by this operation.

Coating or Furring.—A coated tongue is observed either as an independent affection (glossitis) in the course of stomatitis, or as a result of constitutional or organic affections impairing the digestion of food. As soon as mastication and deglutition are interfered with, the automatic self-cleansing of the mouth, which even careful oral hygiene cannot replace, fails; the tongue becomes coated, which means that desquamated epithelial masses and other débris remain there. Considerable cornification of the papillæ filiformes of the lingual epithelium sets in, accompanied by light inflammatory manifestations (hyperemia of the papillæ) as a consequence of defective mastication and deglutition. The coating of the tongue consists of cornified, desquamated epithelia, desquamated tips of papillæ, leukocytes, mucus, lime and cholesterol crystals, bacteria, mycelia, food remnants, and a brownish coloring substance. The coating usually is yellowish-white to yellowish-brown. Colored articles of food (red wine, blackberries, cocoa, etc.) often render the color intense.

Coating of the tongue occurs not only in grave febrile constitutional diseases (typhoid, sepsis, etc.), but in all conditions in which mastication and deglutition are impaired (hemiplegia, gastrointestinal affections, etc.). The appearance of a coated tongue is sometimes, but by no means always, a sign of poor appetite and nutrition. When there is no disorder of the general condition, a coated tongue must be regarded as an isolated catarrh of the tongue (glossitis).

If patients keep their mouths continually open in the course of the affections mentioned, the cavity will become dry and the coating of the tongue turn brown. The surface of the tongue may become fissured and fragile, and slight hemorrhages tend to impart a pronounced brown color to the coating.

Treatment.—Deodorizing and weak antiseptic mouth-washes, to modify and remove the coating, should be used, but no highly astringent or caustic agents. Exaggerated mechanical cleansing of the tongue by scraping or brushing is usually unnecessary and not apt to improve conditions for any length of time. In constitutional diseases, proper oral hygiene may prevent the occurrence or increase of the coating (see page 296).

Lingua Geographica.—The geographic tongue is an affection of childhood and occurs until the advent of puberty. It is rare in later years. There are round, intensely red plaques on the tongue which vary in form and size and are often protruding. There is often a sharply demarcated gray marginal zone consisting of small, dense gray patches—enlarged papillae filiformes with thickened epithelium. The papillae fungiformes are likewise thickened, enlarged, and intensely red. A characteristic sign of these plaques is their inconstancy. They are capable of considerably changing their size and form within a short time; they blend or disappear, while new ones appear in other places. Occasionally there is simultaneous diffuse stomatitis. In other cases rhagades are formed which may cause pain, while the affection otherwise gives rise to no complaints. Attention has latterly been called to the fact that this affection is very often met with in children with an exudative diathesis, from which the conclusion has been drawn that the measures employed for the improvement of this diathesis and other disorders of nutrition in childhood are apt to modify or remove this affection. It may occur as a family affection, and is often congenital. Microörganisms have so far not been discovered as exciting factors. The geographic tongue is not infrequently found in nurslings. It is a permanent affection in that it persists until puberty, when it disappears. It is thoroughly benign and has nothing to do with syphilis, though such a relation is often assumed.

Treatment.—The affection itself requires no treatment, but any coincident or causative disorders of nutrition or tendency to exudation should be treated. Careful dental hygiene is of the greatest importance (see page 294).

Hair-tongue (Lingua nigra).—This affection is always located on that part of the dorsal surface of the tongue which is situated immediately in front of the papillae vallatæ. At this spot there is a peculiar discoloration, of varying hue and intensity; yellow, brown, black and green hair-tongues have been described. The affection is found in an irregular, oval or triangular area. Its peculiar appearance is due to the fact that the papillae filiformes are considerably elongated and thickened, and occasionally assume the appearance of bristly thick hairs. Stomatitis and excessive smoking seem to be capable of causing it. The black or brown color is due to the cornified epithelia. The affection causes no discomfort in the mouth except a mushy sensation and a stale acidulous taste. The patients, however, often suffer from gastro-intestinal disturbances. The duration of the condition varies.

Treatment.—Treatment is really unnecessary. To soften the cornified masses, salicylic acid, resorcinol and hydrogen peroxid are useful. The affected parts may, if necessary, be first scraped. It should be remembered that cases have occurred of hair-tongues making their first appearance after the use of hydrogen peroxid.

PHLEGMONOUS PROCESSES OF THE TONGUE.

These occur in the form of superficial or deep abscesses or diffuse inflammation of the lingual substance (glossitis acuta diffusa).

Abscess.—The usual seat of lingual abscesses is at the dorsal surface of the tongue, toward the base. They originate from bacteria in the mucous glands and follicles of the tongue or from small foreign bodies which have penetrated into the tongue. The abscesses develop gradually, usually with an accompaniment of moderate pain. The swelling of the affected part interferes with swallowing and speaking. As a rule the abscesses are situated in one side, rarely in the middle of the tongue. They may attain to a considerable size, and can be diagnosed from the tense condition and the sensation of fluctuation.

Treatment.—Incision and evacuation of pus will lead to a prompt cure.

Acute Diffused Glossitis.—Deep phlegmons of the tongue are rare, and occur in the wake of grave stomatitis (mercurial) and of infectious constitutional diseases (typhoid, erysipelas, variola, anthrax, etc.). Insect bites and penetrating foreign bodies may likewise cause phlegmons of the tongue. Among the symptoms are high fever and considerable swelling of the tongue, which may reach such proportions that the mouth is too small to hold it. There are violent pains, radiating into the ear. The surface of the tongue is dark violet or bluish-red. The edema may extend to the fauces, palatal arches, and entrance to the glottis. The affection is always a very severe and serious one, especially because there is danger of general pyemia with fatal issue. On the other hand, the inflammatory processes may spontaneously disappear. An abscess in the deep tissues of the tongue affords better opportunity for successful treatment than one that is superficially located. Abscesses are readily formed in the loose connective tissue between the geniohyoglossus, hyohyoid and hyoglossus muscles. No doubt it is often exceedingly difficult to recognize a deep abscess; indeed, this may be impossible without exploratory puncture and incision. Chronic deep lingual abscesses are said to occur which run a very slow course, with comparatively favorable prognosis.

Treatment.—If the initial course of the affection is mild enough to justify the assumption that no abscess will be formed, the treatment may be confined to the application of ice and cooling antiseptic mouth-washes. The same treatment would be indicated in hopeless cases (*anthrax*). In grave cases and in the presence of dyspnea, tracheotomy is advisable in order to prevent sudden suffocation. If an abscess is probable, the therapeutic endeavors should be directed to opening it with all available means. Evacuation of the abscess materially improves the prognosis, as it tends to rapidly reduce the inflammation. The incision is to be kept open. In

the absence of an abscess, scarification of the tongue longitudinally may be effected in serious cases. The wound fissures are tamponed with iodoform gauze or rubbed with iodoform mass (page 298). Hemorrhage, which is usually considerable, is soon arrested.

Decubital Ulcer of the Tongue.—When the tongue is exposed to constant friction by carious or inward growing teeth, or by sharp edges and rough places, its exposed part is deprived of epithelium, and the underlying tissues undergo inflammatory induration. There is at first a small nodule, painful during speech or mastication, which gradually grows and may reach the size of a hazelnut. With increase in size the painfulness increases. The surface of the nodule may disintegrate, forming an ulcer. Both induration and ulcer may heal spontaneously when the roughness has been polished off by the tongue. The affection itself is harmless, but, as it may be mistaken for carcinoma, its diagnosis is important. Indeed, neglected decubital ulcers of this kind may occasionally develop into carcinoma. The diagnosis is very easy in view of the pronounced tendency of decubital ulcers to heal after the offending tooth or other exciting cause has been removed.

Treatment.—Irregularities of the teeth, if any, are corrected, or the offending teeth extracted. The ulcer heals in from eight to ten days.

Chronic Superficial Glossitis.—Moeller, in 1851, was the first to describe this affection. It is a peculiar inflammation of the lingual surface, occurring mostly in females. There are severe burning pains which may become insupportable in mastication or prolonged speaking, and irregularly disseminated red patches and striæ on the dorsal aspect of the tongue and especially at the tip. The epithelium around the patches is very thin or slightly defective, and the mucous membrane underneath shows small-celled infiltration. The papillæ in the area of the inflamed parts are often hyperemic. The foci are, as a rule, found on the dorsal surface of the tongue, at the tip and the lateral margins, the other parts appearing normal. The pains are strikingly severe, considering the slight anatomic changes—so much so that ingestion of food may have to be restricted to the utmost. Should this occur in undernourished patients, severe disorders of nutrition may follow. The course is very chronic. The affection develops in sudden spurts, alternating with comparatively quiescent periods lasting for weeks or months. In this way it may persist for years.

Treatment. Painting of the affected parts with silver nitrate and lactic acid has sometimes proved beneficial. Rinsing with a blackberry decoction, and restriction to a salt-free and non-albuminous diet, have been recommended.

Acute Papular Glossitis. All the cases of this rare affection so far described have occurred in women. It seems to commence with slight febrile manifestations, lassitude and anorexia, followed by

burning pains in the tongue and the formation of isolated white patches on its surface the size of a pea. These patches become erosions with red and serrated edges, the base covered with pus. The affection seems to heal spontaneously, after having continued for about three weeks. Nothing is known as to its etiology. The foci resemble, to a certain extent, variolar and varicellar pustules.

Treatment.—Oral hygiene, antiseptic mouth-washes (page 296).

Macroglossia.—Abnormal enlargement of the tongue occurs congenitally, its development in childhood or later being rare. The enlargement may attain to the proportions of elephantiasis. Macroglossia is usually due to lymphangioma, although there is also a muscular form in which the tongue attains to twice or three times its normal size, or it may acquire abnormal length, though covered by normal mucous membrane. This form is always congenital. Ingestion of food being hindered, operative intervention in the shape of cuneiform excisions may be necessary. Ligation of the lingual artery has also been recommended. Macroglossia is sometimes a manifestation of general acromegaly. The lymphangiomatous form is also found in cretinism.

Lingua Plicata.—Related to congenital macroglossia is the furrowed tongue. The tongue is enlarged, though not extremely; its shape is approximately normal, but its surface is covered with numerous symmetrically arranged furrows, the deepest being in the median line. The prominent parts have normal papillae, while in the furrows there are none. Neurasthenic individuals may suffer much inconvenience and pain from this condition. The plicated tongue not infrequently develops into the geographic form (page 325).

AFFECTIONS OF THE LINGUAL TONSIL.

The follicular structures at the base of the tongue are called lingual tonsils.

Acute Lingual Tonsillitis.—This acute inflammation frequently occurs in inflammation of the palate and parenchymatous tonsillitis. The follicles are reddened and the region of the lingual tonsil is swollen. There is no need for special treatment.

Hypertrophy of the Lingual Tonsil.—The swelling affects either isolated follicles or the entire tonsil, the superficial veins being varicosely dilated. These veins may even burst during coughing or vomiting, giving rise to an alarming though harmless hemorrhage. The hypertrophy is apt to impart a guttural sound to the speech, interfere with singing, and cause fatigue and cough. It also gives rise to a feeling of oppression in the mouth, simulating the presence of a foreign body.

Treatment.—Cauterizing agents and similar remedies are usually unsuccessful. Should energetic measures be indicated, the entire hypertrophic tonsil should be ablated.

Hyperkeratosis.—This affection is often located on the hyperemic lingual tonsil and is an abnormal process of cornification, forming in the crypts of the lingual tonsil hard hair-like cones which may protrude from the crypts like hairs and can be removed with forceps. Often there is no subjective sensation whatever except scratching and pressure, and occasionally an unpleasant, putrescent taste in the mouth.

Treatment.—The affection often disappears spontaneously, and requires no treatment unless the symptoms are severe. If any eradication measures are resorted to, they should be energetic—curetting and the Paquelin cautery.

DISEASES OF THE SALIVARY DUCTS.

Sialodochitis.—Inflammation of the excretory ducts of the salivary glands may be due to invasion by bacteria or the introduction of foreign bodies into the excretory ducts. The submaxillary duct is most subject to attack; it terminates in an aperture at the salivary caruncle which is permeable by a thin sound, while immediately below the aperture it is conically enlarged, and beyond this again, for two-thirds of its extent, it is narrower. Foreign bodies are therefore apt to be arrested at the anterior enlargement of the duct.

The sublingual gland usually terminates in several short ducts, thus offering difficulties to the entrance of foreign bodies. The terminations of the anterior lingual glands present a similar structure. The aperture of the parotid duct is comparatively large, but, as there is a decided curve immediately behind it, foreign bodies are hindered from entering.

Inflammation will result either when foreign bodies are arrested in one of the ducts or when there is a bacterial infection without foreign bodies. The ducts then exude a purulent secretion which can be expressed between the fingers; later on, a large quantity of viscid saliva may often be evacuated which has probably been retained in the ducts by swelling of the mucous membrane. Frequently there is periodic spontaneous evacuation, as, for instance, while eating, or even at the mere sight of food. This may be accompanied by severe pain in the buccal fundus (*coliques salivaires*), and occasionally by a disturbance of the general condition, chills and fever. These painful paroxysms often occur at long intervals, but, later, follow in more rapid succession. Finally the evacuation of pus occurs during the painless intervals also (*pyorrhea salivaris*).

Sialoliths.—Impacted foreign bodies may develop into salivary calculi by incrustation, but these may also be formed without the presence of foreign bodies. In any case, the formation of stones can occur only in the presence of sialodochitis. Stones are least

frequently formed in the parotid and sublingual ducts. The submaxillary duct is most often affected. The stones are located 1 or 2 centimeters behind the sublingual caruncle, and are elongated, pear-shaped, or, less often, round. They vary in size from a grain of wheat to a date kernel, but may in exceptional cases become much larger; stones the size of a hen's egg have been observed. In most cases they are grayish-white and of mortar-like consistency, though sometimes they are harder and darker. They consist of calcium phosphate, less often of calcium carbonate. Should there be any marked inflammation at the same time, the walls of the distended salivary duct may be destroyed by ulceration, thus embedding the stones in an abscess. The circumstances may be such that the pus perforates outward, causing the formation of a fistula which will not close until the stones are removed.

Symptoms.—The symptoms of salivary calculi resemble those of simple inflammation and foreign bodies in the duct, and consist of pyorrhea, swelling in the vicinity of the duct and the glands appertaining thereto, and pain. The diagnosis is made with the sound and is particularly easy in the submaxillary duct. Stones in the parotid duct may have to be demonstrated by bimanual palpation. Pathologic thickening of the floor of the mouth should always raise the suspicion of salivary calculi. Their presence is sometimes revealed by the Roentgen ray (see Plate XXII, Fig. 4).

Treatment. If possible the stone should be removed, and this can in nearly all cases be done under local anesthesia. If the stone is embedded in an abscess, it can be easily removed after incision of the latter. If the stone cannot be detected after incision of the duct, it may be expelled spontaneously after a few days. Should a stone lie in the gland itself, the gland had better be extirpated *in toto*.

DISEASES OF THE SALIVARY GLANDS.

Secondary Sialadenitis in Affections of the Salivary Ducts.—

If sialodochitis has developed from foreign bodies, infection, or salivary stones, the salivary glands affected frequently undergo rapid swelling. The skin over the gland is hyperemic and sensitive, but the swelling usually subsides promptly without causing an abscess. Should this occur often, it will lead to dilatation, induration, and slight painfulness of the gland from inflammatory infiltration of the glandular connective tissue, accompanied by slight pain in deglutition both in the throat and in the region of the gland. On the other hand, the whole trouble sometimes rapidly disappears, even after repeated swelling of the gland.

Treatment.—Bimanual massage of the glands, painting with iodine, and the administration of salicylic preparations are advisable. Should dilatation and painfulness persist, the gland should be extirpated.

Salivary calculi in the glands are usually multiple, and from the size of a millet seed to that of a pea. Should they persist until the gland is chronically affected, the latter should be extirpated.

Diseases of the Salivary Glands in General Affections.—The salivary glands, especially the parotid, are usually involved when the patient is suffering from septic affections and grave infectious diseases (scarlatina, measles, variola, typhoid). Suppurative inflammation in these conditions is prognostically unfavorable; it usually leads to an abscess which has to be incised, preferably from without. Ulcerous parotitis not infrequently leads to general septic infection. Inflammatory changes in the salivary glands are always present in rabies.

Inflammation of the salivary ducts after surgical operations is likewise a prognostically unfavorable condition. Probably it originates in the mouth, especially when the amount of ingested food has been small and the mouth is poorly cleansed. Here, again, an abscess is liable to develop, leading to general sepsis.

There are also inflammations of the salivary glands without septic or infectious general disease and without involvement of the salivary ducts, in both adults and children. In adults the parotid is often involved, in children the submaxillary gland. These glandular affections are often symmetric and accompanied by moderate pain, slight elevation of temperature, swelling of the glands, and some disturbance of the general condition; they may also lead to abscess. Nothing definite is known in regard to their etiology.

Actinomycosis, Syphilis, Tuberculosis.—*Actinomycosis* of the salivary glands is rare as a primary affection, while as a secondary involvement in actinomycosis of the oral cavity it is more frequent. *Syphilis* of the salivary glands occurs in the form of gummatous enlargement of the affected glands, but up to the present it has been observed only in the sublingual and anterior lingual glands. *Tuberculosis*, as a rule, affects only the parotid gland.

Epidemic Parotitis.—This is the result of a contagious affection, the frequent involvement of the testes and pancreas pointing in that direction. The parotid gland is infected from the mouth, and the parotitis is frequently accompanied by slight stomatitis. The affection runs its well-known course, with symmetric swelling of both parotids, hyperemia of the skin, light fever, moderate interference with the general condition, painfulness of the glands, and occasionally simultaneous swelling of the neighboring glands. As a rule it subsides within a week. It may, however, be followed by otitis, a possibility which should be given due consideration.

Treatment.—An affected child is best isolated. If there is fever, the patient is put to bed. To relieve the painful tension over the glands, warm oil or petrolatum is applied to the swollen parts and covered with cotton, or an application of aluminum acetate solu-

tion is made. Frequent rinsing of the mouth and gargling with a 2-per-cent. solution of hydrogen peroxid is advisable to prevent further infection. Should the swelling not readily subside, unguentum citrinum may be rubbed in. In case of ulceration, an incision will be necessary. The diet should be fluid or pappy. Care should be taken to maintain regularity of the bowels. Complicating orchitis is treated by elevation of the testicles and cold compresses.

Chronic Enlargement of the Salivary and Lacrimal Glands (Mikulicz's Disease).—This rare affection is characterized by symmetric and simultaneous involvement of the lacrimal and large salivary glands. The sublingual, lingual and palatal glands may or may not be simultaneously involved. The characteristic symptoms consist of swelling of the glands referred to, which imports a striking, characteristic appearance to the face. The etiology is by no means clear. There are simple cases in which only the glands mentioned are affected, and others in which the lymph glands and the spleen also are swollen, and others again presenting leukemic and pseudoleukemic blood changes. True hypertrophy of the glands seems also to occur.

Treatment.—Therapeutically, Roentgen-ray treatment has been successful.

Ptyalism.—Excessive secretion of saliva occurs as a concomitant manifestation of inflammatory affections of the oral mucous membrane, in mercurial poisoning, and as a nervous affection. The treatment consists in removal of the cause and restriction of the secretion by belladonna preparations.

Aptyalism.—This condition occurs in nervous affections, rarely in inflammatory ones. It is associated with an unpleasant sensation of dryness in the mouth. Pilocarpin, 0.005 to 0.01 Gm. ($\frac{1}{7}$ to $\frac{1}{2}$ grain), subcutaneously or by mouth, may be tried, to incite secretion of saliva. Painting of the oral mucosa with glycerin may relieve to some extent the sensation of dryness.

PHLEGMONS OF THE BUCCAL FUNDUS.

Ludwig's Angina.—This purulent affection occurs in the loose vascular and lymphatic connective tissue of the buccal fundus in which the salivary glands are embedded. This tissue becomes infected through disease of the buccal fundus, after operations and injuries, or through carious teeth. The pathologic picture usually develops very rapidly, a hard distention of the region between the inferior maxilla and the hyoid bone occurring inside of a few hours. This rapidly increases and is accompanied by considerable edematous swelling of the skin of the throat and chin. The inflammatory swelling in the interior of the mouth extends to the deeper connective tissue of the neck, the entrance of the

glottis, and sometimes to the mediastinum. It soon produces difficulty in breathing. The head is carried stiffly, with the chin raised, and considerable pallor and pronounced cyanosis rapidly make their appearance. The patient can hardly open his mouth, and inspection of the cavity is difficult. The condition must therefore be ascertained by palpation. The tip of the tongue is found raised, often closely pressed against the hard palate by the swollen tissues underneath. In this way the affection soon assumes an extremely dangerous and tormenting aspect—dangerous owing to impeded respiration and possible general septic infection, with chills, fever, icterus, albuminuria, and endocarditis. General sepsis is often the cause of death, and the prognosis is therefore very doubtful. The most favorable course is the rapid evacuation of the abscess, the pus perforating through the mucous membrane of the buccal fundus; but this is a rather rare occurrence.

Treatment. The possibility of rapid extension demands energetic surgical intervention. Even before there is any demonstrable pus, free incisions must be made in every case, in order to decrease the tension of the infiltrates by evacuation of blood and exudates. The incision is best made close to the submaxillary margin, to avoid the sublingual artery, the lingual nerve, and the submaxillary duct. Injuries to the sublingual gland are unimportant. The incisions should be tamponed to prevent too profuse hemorrhage. The galvanocautery can be used for incising, but in any case the incisions must be made at great depth, because the pus is often located as deeply as beneath the sublingual gland.

AFFECTIONS OF THE ALVEOLAR PROCESSES.

Parulis (Periostitis alveolaris dentalis).—Gum-boil is often associated with carious teeth. It spreads from the carious points, through the pulp to the apertures of the dental root canals, creeps along the alveolar margin and continues through the alveolar wall or through the osseous canals to the periosteum of the alveolar process. In this way an abscess is formed underneath the maxillary periosteum, usually in the vicinity of the apex of the dental root. This is accompanied by considerable edematous swelling of the gums, finally leading to much swelling of the adjacent soft parts of the cheek. The entire process may run a very acute course, in which case it is accompanied by severe pains, especially when the tooth is involved. The pains are somewhat abated as the abscess is being formed. This acute and very painful form is often found when the pulp is not freely exposed, and so the inflammatory products are arrested in the pulp cavity. If, on the other hand, the latter is wide open, allowing pus and inflammatory products to escape, the course may be less acute, with pain from mastication or other pressure only. Patients have a sensation of the teeth being too long,

an indication that they are not firmly held in the alveoli. Small or large pus pouches or granulations are often formed at the apex of the roots. By this means a chronic periodontitis may be changed to alveolar periostitis, or parulis. In the acute form of parulis the general condition is often considerably impaired by fever and headache. The abscess being formed, the pus soon burrows toward the gums, usually toward the malar side of the alveolar process, and at the upper lateral incisors toward the palate or buccal fundus. Under certain circumstances this may lead to palatal abscesses, which are always located laterally. At times the perforation does not take place immediately, but assumes the shape of a fistula, the so-called dental fistula, which may persist for a long time. (Plate XXIII.) The pus rarely perforates through the outer skin; when it does, the point of exit is most frequently at the posterior molar teeth of the inferior maxilla. Perforation toward the buccal fundus may cause genuine Ludwig's angina, rendering the diagnosis difficult (see page 330).

Treatment.—Abscesses are incised, preferably parallel to the alveolar margin. A useful measure is to keep the wound open by an iodoform tampon. Above all, the affected tooth is removed or adequately treated, in order to prevent a recurrence. It is then left for the dentist to continue the correction of the teeth. Wisdom teeth are best extracted, because they do not readily lend themselves to other treatment owing to their hidden position. Alveolectomy may be done in selected cases.

Pyorrhea Alveolaris.—The accumulation of tartar about the neck of the tooth favors the development of pyorrhea alveolaris, or Rigg's disease. The adjacent gums become swollen, with a red or blue-red edematous appearance. As the gum margins detach themselves, pouches and crypts are formed, containing soft, easily bleeding granulation tissue and slimy detritus, with large quantities of endamebæ, spirochetæ, and bacteria. These masses can be expressed by pressure upon the gum. At times pus also is evacuated. It is possible with a thin sound to uncover the neck of the tooth for a considerable distance. The affected gums bleed easily. The affection is at first almost painless, and no attention is paid to it for a long time. Gradually the tooth becomes loose, causing pain in biting and masticating. The incisors and the upper anterior molars are attacked first. As a general rule the teeth of the upper maxilla are affected sooner than those of the lower. In most cases single groups of teeth are attacked at one time, rarely all the teeth at once. The diagnosis in doubtful cases is easily confirmed by Roentgen-ray examination.

Treatment.—The first step is removal of the accumulations of tartar by the dentist. Pockets of any appreciable depth are freely incised with the knife or galvanocautery, each tooth being treated separately. The pockets are then rubbed with iodoform mass (page

298) by means of a cotton-covered sound. Deeper pockets may be tamponed with a piece of iodoform gauze. This simple treatment will often effect a cure in a few weeks in light cases, the incisions being frequently repeated should the cut edges agglutinate. If the swelling of the gums persists, it is advisable to cauterize the margins with the galvanocautery or the silver nitrate stick. As pyorrhea is often found in *tabes* and *diabetes mellitus*, attention must be directed to these diseases as well as to other constitutional ailments. Emetin given by subcutaneous injection or orally affects only such endamebæ as are within the reach of the blood; those on the surface of the mucous membrane seem not to be destroyed. The mucosa itself possesses the ability to prevent their migration through it. When emetin is used in the treatment of pyorrhea, two drops of fluidextract ipecac in a tumbler of water, used as a mouth-wash, will hasten the cure.

Mercuric succinimide injected intramuscularly in doses of 0.06 Gm. (1 grain) at weekly intervals, until six injections are given, yields gratifying results in many cases of pyorrhea alveolaris. The mercury salt is dissolved in 1 Cc. (15 minims) of hot sterile distilled water and injected in the manner described on page 581.

Attempts have been made to treat alveolar pyorrhea with bacterial vaccine. Several different organisms are usually present, and an autogenous vaccine should be made up for each case. These vaccines are often of great value. A stock vaccine containing cultures of the organisms usually present in pyorrhea cases is also to be had, and good results are reported from its use.

The teeth should in no case be extracted so long as they are firmly embedded; even loose teeth become firm again under appropriate treatment.

In endamebic pyorrhea, constitutional conditions secondary to this infection often occur, the most frequent being arthritis of the so-called deformans type. The combined local and systemic treatment with emetin brings about recovery in a large percentage of cases (see page 723).

Gingivitis (Inflammation of the gum).—Mechanical factors, such as the ingestion of too hot foods, improper ventilation, mouth breathing, and gum chewing, enter into the etiology of this condition, as well as bacteria.

AFFECTIONS OF THE LIPS AND CHEEKS.

Congenital Fistulæ of the Lower Lip.—Fistulæ are met with not only in cases of hare-lip, but in otherwise normal lips. Sometimes they constitute a family affection. They are usually bilateral and are symmetrically located about one centimeter from the median line, forming fine ducts up to one centimeter deep which run posteriorly and inferiorly toward the mucous membrane.

They originate in a semilunar excavation in the red of the lips, which is often present in subjects free from fistula. Mucous secretion is evacuated through the external orifice, and this is the only inconvenience occasioned by the fistula, which is probably due to incomplete closure of embryonic furrows.

Acute Cheilitis.—Acute inflammation of the lips often follows in the wake of injuries or other affections of the lips (herpes labialis, eczema, etc.). The lips may become considerably swollen, forming probosciform eminences. Acute cheilitis will, of course, also occur when a *furuncle* or *carbuncle* appears at the lips. This often leads to enormous edematous swelling of the lips, accompanied by great pain. Labial furuncles and carbuncles are often the cause of general infections. The possibility of an *anthrax pustule* should always be thought of when a labial furuncle threatens to assume a malignant course. The anamnesis and bacteriological examination will then establish the diagnosis. An *abscess of the lips* occasionally develops from infected glands of the mucous membrane. It is located rather deeply in the tissue underneath the mucosa and may lead to great pain and swelling.

Treatment.—The treatment depends upon the underlying disease. Abscesses must be incised. Anthrax pustules are to be excised only in the very beginning; after the process has extended further, a waiting attitude is indicated, the more so as the spontaneous cure of anthrax pustules is no great rarity.

Chronic Cheilitis.—*Cheilitis Glandularis.*—This affection is always associated with swelling of the lower lip, without causing any particular complaint until the lip finally becomes much swollen, firm and immovable. It originates in the mucous glands, which may become enlarged to the size of a pea. The excretory ducts are dilated and secrete viscid mucus or purulent fluid. Ulceration of the swollen glands follows, a small abscess forming which perforates toward the oral cavity. Mucus and pus continue to be evacuated at the place of perforation for a long time.

For internal treatment, potassium iodid is recommended. The abscesses themselves are incised or cauterized, after which they are painted with tincture of iodine.

Cheilitis exfoliativa is located at the lower lip. The mucous membrane is intensely red, completely loses its epithelium in spots, and becomes loose. The affected lip is exceedingly painful, causing the patient to continually press it outward to avoid contact with the teeth. In this way the ailment becomes very tormenting. It may last several weeks or months, but appears to be subject to spontaneous cure.

There is no special treatment for this affection. Covering the affected labial mucosa with mild salves or boric acid ointment, spread on a small piece of linen, gives material relief.

AFFECTIONS OF THE MALAR MUCOSA.

The malar as well as the labial mucosa is subject to glandular disorder. The lymph glands located in the cheek are not infrequently affected, and may swell in tonsillitis and coryza.

BENIGN TUMORS OF THE MOUTH.

The mouth may harbor all known forms of tumors, some of them with especial frequency.

Fibroma.—When pure fibroma occurs in the mouth, which it rarely does, it is most frequently found upon the surface of the tongue and has a more or less pediculated form; occasionally fibromata are found deeply embedded in the lingual substance. The tumors are usually very hard. Neurofibromata likewise occur in the tongue. The superficial tumors may, as a matter of course, exhibit the most varied forms and sizes. The important point is that they are always sharply demarcated from the surrounding tissue. Their slow growth is characteristic. As a rule they cause but slight discomfort until they attain considerable size. The surface of the soft tumors may under certain circumstances become ulcerative. Lingual fibroma is met with congenitally in infants and at any later age. The diagnosis is not easy when the tumors are soft, especially when they ulcerate and bleed. In that case syphilis should be taken into consideration. Carcinoma is in most cases distinguished by its characteristic appearance. The differentiation from sarcoma is a matter of great difficulty in certain cases.

Treatment. When, in doubtful diagnosis, potassium iodid and mercury have failed, the alternative is operation. Fibromata can always be very easily enucleated under local anesthesia. Should enucleation be difficult, the best plan is to make an exploratory excision for microscopic examination, further surgical steps to depend upon the findings. The application of the Roentgen ray gives good results.

As stated, fibromata are very rare in other parts of the mouth. They have been described as occurring in the hard palate, at the uvula, palatal arches, interior surface of the upper lip, and the malar mucosa.

Lipoma.—The tongue is most frequently the seat of oral lipoma; fibrolipoma usually occurs singly. In most case lipomata are located immediately under the surface, rarely intramuscularly. They are usually smooth to the touch, of roundish shape, and may attain to the size of a hen's egg. In making the diagnosis, gumma of the tongue has to be considered. Lipoma has been observed at the fundus of the mouth, where it can give rise to symptoms by pressing the tip of the tongue upward; in that case the diagnosis

may be more difficult. There are also lipomata of the cheek, but the lips, gums and palate are very rarely attacked.

Treatment.—Lipomata can always be easily removed by surgical operation.

Myxoma. Myxomata in the mouth are extremely rare. A pure myxoma has been observed on the malar mucosa. These growths occur occasionally at the point of fusion between the hard and the soft palate.

Myoma.—Myomata are very rare.

Chondroma, Osteoma.—These growths occur at the soft parts of the mouth, but very seldom. Lipo- and fibro-chondromata have also been observed.

Hemangioma. *Telangiectasia* (angioma simplex) consists of a clump of minute bloodvessels and occurs at points where embryonic fissures have undergone fusion. It appears as a flat, slightly gibbous, sharply but irregularly demarcated red or blue-red tumor. The majority of these little tumors are congenital. Their favorite site is the lips, especially at the external surface and the red part. When these growths become larger they develop into cavernous angiomas.

Cavernous Angiomata.—These are tumors of various sizes consisting of numerous bloodvessels which form dilated thin-walled cavities of spongy tissue. The simultaneous appearance of telangiectasia and angioma cavernosum is not infrequent. Angiolipoma and angiosarcoma are also to be considered. The cavernous angioma has a bluish color, a gibbous surface, and a flabby feel. It can easily be evacuated by pressure. It occurs at all points in the mouth, but most frequently in the tongue. It is often multiple, notably when located at the anterior part of the tongue. These growths may attain to considerable size and occasionally spread to the buccal fundus. They have also been observed at the cheeks and lips. They are really benign tumors without metastases, but have often a great tendency to extend to adjacent tissues. They may also give rise to serious hemorrhages. Furthermore, they are associated with light inflammatory manifestations which involve the danger of general septic infection.

Treatment. In consideration of these dangers the removal of the tumor is very desirable. This can only be done by surgery.

Racemose Aneurysm.—Racemose aneurysm occurs almost exclusively in the head and represents the well-known dense plexus of largely dilated, tortuous, communicating arteries. The tumors of the head and face may occasionally spread to the oral mucosa and form large tumors. This can most readily happen with aneurysm of the internal maxillary artery, where the newly formed vessels extend to the labial mucosa, gums and palatal arches, giving rise to hemorrhages which can only with difficulty be arrested.

Treatment is exceedingly difficult and at times almost impossible.

Lymphangioma.—Lymphangiomata often occur in the mouth, particularly at the tongue in the shape of verrucous, nodiform, diffuse and aplastic growths. They are probably neoplasms of lymph vessels and lymphoid tissue of congenital origin. They are usually situated at the location of closed embryonic fissures. They can often be observed at the earliest age, and remain small for a long time. As a rule they increase in size intermittently in connection with the occurrence of inflammatory processes of the tumor tissue. Inflammations of this kind are easily caused by topical injuries. Coincidentally there may be considerable swelling of the tongue or pronounced glossitis. These inflammatory processes always involve the danger of infection of the cervical connective tissue and edema of the glottis.

Nodiform and verrucous lymph glands occur at the margins or dorsal part of the tongue in the shape of coarse tumors of varying size, the uneven surface of which resembles lingual papillae. The small eminences on the tumor, however, are not true papillae, but rather vesicles containing fluid, which can often be recognized as such macroscopically. A large number of small foci can thus be formed on the tongue in an irregular, diffuse way, or even large coherent tumors. Just at the time when a glossitis is newly developed, an entire half or the whole of the tongue is enlarged, hyperemic and painful, and the real cause of the glossitis may be entirely overlooked.

Diffuse Lymphoma.—It has already been mentioned that macroglossia may be caused by lymphoma formation. There are small, fluid-containing vesicles under the transparent surface of the lingual mucous membrane, which are diffusely disseminated over the larger part of the tongue, not only on the surface, but also in the lingual substance. As a result the tongue may become so large that part of it will protrude from the mouth and be thereby exposed to a variety of injuries, infections, and desiccation. The enlarged tongue may cause displacement of the teeth, and the entire submaxillary bone may become deformed.

Cystic lymphoma is exceedingly rare, and is usually situated at the anterior part of the tongue; it may attain to the size of a lemon. It also occurs at the lips, especially the upper one, the cheeks, and the buccal fundus. Inflammatory manifestations may also, though rarely, be associated with this growth. The formation of vesicles is a characteristic diagnostic point.

Treatment.—Small flat lymphomata of the first named variety may be left undisturbed, unless they cause discomfort. The larger tumors will have to be removed surgically. The operations should be carried out at a time when there are no inflammatory manifestations. Good results have been attained with the cautery.

Dermoid Cysts.—Dermoid cysts do not often occur in the mouth. When they do they are found almost exclusively at the fundus,

either exactly in the center or close to the median line. The medial cysts are distinguished from the submental and the sublingual. They originate in all cases from an epidermal cell caught in the closure of embryonic fissures. They are mostly found densely arranged at and behind the medial part of the inferior maxilla or the hyoid bone, a fact which likewise points to embryonic origin. The sublingual dermoids belong entirely to the buccal variety; they bulge out considerably in the region of the frenum lingue, raise the tip and posterior part of the tongue, and, as they grow, may cause in this way considerable trouble in swallowing and speaking. The submental dermoids, when greatly developed, may cause a kind of double-chin, together with serious difficulty in swallowing and breathing. Since the cysts grow very slowly, patients usually get accustomed to them so that no notice is taken of them until a relatively late stage. Inflammatory processes and trauma may cause a rapid increase in their growth. The buccal aspect of a sublingual dermoid is spherical or oval, with a smooth and movable investing membrane. The walls are usually very thick. Pressure upon the cysts may leave an impression. Bimanual palpation will serve to determine the exact position. Movement of the tumor in swallowing indicates its firm adhesion to the hyoid bone.

Treatment.—Enucleation is therapeutically the most desirable procedure; it is easy in all cysts which have not passed through any inflammatory process. In the new-born who cannot nurse well, owing to cysts, puncture may perhaps suffice, enucleation being postponed to a later period. Excision of only part of the cysts, followed by irritation, or puncture, with subsequent injection of tincture of iodine, is to be deprecated, since these procedures may easily lead to inflammatory processes in the neighborhood of the cysts.

Cysts Originating from Glands.—Cysts in the mouth are often found, the walls of which are invested with glandular epithelium, and which contain fluid or mucous masses.

Superficial cysts of the mucous membrane are probably the result of the excretory ducts of the glands having been occluded by inflammatory processes. The cysts are most frequently found where there is the greatest accumulation of glands—the inner surface of the lips, the malar mucosa, the dorsal surface of the tongue, especially in the region of the papillae vallatæ, and the inferior surface of the tongue. They have only rarely been observed at the uvula. The size of the cysts varies from a millet seed to a pea. Their walls are usually very thin and transparent. The subjective symptoms are very slight in most cases, and often do not arise until the appearance of inflammatory manifestations.

Treatment.—Enucleation of the cysts under local anesthesia.

Ranula.—Cysts of the buccal fundus are due to enclosure of epithelial elements in the palatal fissures, or to occlusion of excretory ducts of various glands, such as the sublingual, the submaxillary, the glandula incisiva at the neck of the central and lateral incisors, Nuhn's glands in the tongue, and the numerous little mucous glands of the buccal mucosa. Ordinarily it is difficult to say from which gland a ranula emanates. When located precisely in the center, it may be ascribed to epithelial enclosures. In most cases it lies close to the median line, immediately below the mucous membrane. It appears at the buccal fundus between tongue and inferior maxilla as a bluish-red or bluish-white, roundish, transparent tumor, displacing the organs of the buccal fundus to a smaller or greater extent according to its size, and causing discomfort of varying degrees in swallowing and speaking. The diagnosis can readily be made from the presence of fluctuation and by bimanual examination. As a rule the tumors grow very slowly. Infection of the ranula may lead to very unpleasant inflammatory processes in the buccal fundus.

Treatment.—The best treatment in simple cases is the use of a hair rope, *i. e.*, conducting a silk thread through the cyst and tying the ends together. This will serve to gradually evacuate the cyst. The thread is allowed to remain in position for six to eight days, by which time it will have automatically cut through the ligated tissue. Meanwhile the cystic walls may have completely grown together and atrophy commenced, preventing refilling of the cyst. Excision of a piece of the cystic wall may have the same effect. Mere puncture is almost invariably followed by recurrence. Should the hair rope method fail, the cyst should be completely extirpated.

Cysts at the Root of the Tongue are but rarely observed. They may attain to the size of a hazelnut or a small apple. These cysts originate from the superficial mucous glands of the root of the tongue and from remnants of the thyroglossal duct. The latter are embedded either superficially or deeply in the lingual substance; their epithelium is partly of the ciliary variety. When the cysts disturb speech or respiration, or produce the sensation of the presence of a foreign body, operative interference is called for. A hair rope may be applied as in ranula, or a piece of the wall may be extirpated. This proceeding, however, is successful only in unilocular cysts; multilocular ones require as complete an extirpation as possible. Hemorrhage may be considerable.

Struma of the Lingual Base.—The strumæ are flat or semiglobular tumors, smooth or gibbous, of soft consistency, and covered with normal mucous membrane. They are usually situated in the median plane between the hyoid bone, epiglottis and cœcal foramen. They result from remnants of the thyroglossal duct which, of course, is the residue of the epithelial depression from which the thyroid

gland is formed. Lingual strumae, therefore, are accessory thyroid glands. They are very rare in males, and are usually discovered at the time of puberty. As to diagnosis, confusion with cysts of the lingual base should be guarded against.

Treatment.—Serious symptoms from enlargement of the strumae, inflammations, and hemorrhages, may demand operative intervention. Removal of superficial prominent glands is usually easy, under local anesthesia, with the galvanocautery loop. Nodules located deeply in the lingual musculature toward the hyoid bone may have to be removed from without. Attempts have also been made to reduce the tumor by the administration of thyroid or thymus substance. Previous to operation, it should be ascertained whether the thyroid is normal, because in some cases myxedema has been observed to follow operation.

Adenoma. Adenomata of the mouth are very rare. They have been observed at the sublingual gland, the uvula, and the mucous glands of the lips.

Papilloma.—Papillomata are often found in the mouth. They preponderate in the male sex, possibly in consequence of excessive use of alcohol and tobacco, and may perhaps be regarded as a product of chronic irritation. They are usually situated at the uvula and the palatal arches, seldom at the tongue, where they prefer the lingual base. Most papillomata remain small, from the size of a pinhead to that of a pea, rarely exceeding the size of a hazelnut. Owing to their smallness, they cause little complaint. The diagnosis can be easily made from the fine and coarse eminences covering the surface.

Treatment. Papillomata are to be removed with knife or scissors.

Endothelioma. Endotheliomata originate from either the endothelium or the perithelium. The newly formed cells are found in nests and cords in the meshes of a trabecular stroma. The histologic diagnosis is by no means easy at times, especially owing to the similarity of the histologic picture to carcinoma. The connective tissue has a great tendency to undergo a variety of changes, for instance into bone, cartilage, fat, mucous and hyaline degenerations, with the consequence that often there are genuine mixed tumors. Endotheliomata occupy the border-line between benign and malignant tumors, since they are apt to run a thoroughly benign course for many years and then suddenly exhibit a malignant, sarcomatous character.

Their place of predilection is in the most glandular strata of the mucous membrane. They are most frequently found in the hard and soft palates, where the glands are most profuse. Besides, endotheliomata have been observed at the buccal fundus, upper lip, cheeks, and tongue. Palatal endotheliomata are always situated laterally, forming flat or globular, smooth or slightly gibbous eminences, of very hard or tough elastic consistency. They are

covered with smooth, movable mucous membrane. Their size varies from the smallest diameter to enormous dimensions, so that the mouth may be completely filled. Their growth generally is very slow. The surface, being exposed to external injury, may ulcerate. In most cases the tumor is distinctly encapsulated, but the capsule will be perforated as soon as a tendency to malignant growth develops. The clinical diagnosis often presents difficulties in differentiation from other tumors. The subjective symptoms are purely mechanical, as in swallowing, speaking and masticating. Pains accompany ulceration, and when the tumor is located in the palate, they radiate toward the ear.

Treatment.—When the tumors are encapsulated, which is usually the case, they can be easily enucleated through a longitudinal incision. Endotheliomata, even when apparently not malignant, tend to recur after excision.

MALIGNANT TUMORS OF THE MOUTH.

Sarcoma.—The majority of the sarcomata observed in the mouth have been found in one or another part of the tongue. The picture varies considerably, the tumors differing materially as to size, shape and appearance. They may be located in the lingual parenchyma or pedunculated upon the tongue. Ulceration is rare. The largest lingual sarcoma so far observed weighed 400 grams (13 ounces). Microscopically they are usually small round-celled sarcomata, but spindle-celled sarcomata and fibrosarcomata also occur.

Aside from the mechanical symptoms caused by the size of the tumor, sarcoma often causes pains which radiate to the ear of the corresponding side, and which may become exceedingly violent. In most cases the tumor grows relatively slowly; but there may also be sudden advances. Metastases are not frequent; when they do occur it is usually in lymph glands. For this reason, as well as on account of the easy demarcation of the sarcomata, their prognosis is comparatively favorable. Local recurrences often take place after operation, but when these have been attended to a complete cure is sometimes effected. The diagnosis can be assisted by an exploratory excision, or it may be necessary to resort to experimental antisyphilitic treatment. For this purpose large doses of potassium iodid, 4 to 8 Gm. (1 to 2 drams) daily, are administered, which, in the event of a gummatous infection, would have a favorable effect in about two weeks. If the sarcoma is extensively ulcerated, it is almost impossible to distinguish it from carcinoma, but so far as therapy is concerned this is immaterial.

Sarcoma has been found in the palate. A few cases of melanosarcoma in the hard palate have also been described. Myxosarcoma has been observed at the malar mucosa in very rare cases.

Treatment.—As soon as the diagnosis of sarcoma is assured, the tumor should be removed. In doubtful diagnosis but satisfactory demarcation an attempt may be made to enucleate the tumor. In inoperable cases Coley's mixture of erysipelas and prodigious toxins may be injected hypodermically in very small and gradually increased doses; or arsenic, Roentgen ray and radium treatment may be tried, to reduce or remove the growth.

Carcinoma.—Carcinoma of the mouth is of very frequent occurrence. It is a surprising fact that the male sex is preëminently affected. Carcinoma of the tongue and mouth occurs in the proportion of nine men to one woman. The reason for this disproportion is to be found in the use of tobacco. Smoking, rough teeth and the use of too hot drinks are important etiologic factors in mouth cancer. It has already been mentioned that leukoplakia predisposes to carcinoma. Statistical figures of 159 cases of lingual carcinoma, compiled by von Bergmann, show that in about 34 per cent. leukoplakia may be assumed to have been the cause. Chronic labial eczema likewise seems to furnish suitable soil for the development of carcinoma. The ultimate causes have not yet been discovered. Primary carcinoma almost always occurs singly.

Carcinoma of the Lips is oftener of the lower than of the upper lip, the proportion being about 17 to 1. It is mostly located laterally, but its onset almost invariably escapes notice. The first changes attracting attention are usually small, flat indurations in the red of the lips, in the vicinity of the skin border. This indurated spot loses its epithelium, voids some secretion, and bleeds easily. The small tumor gradually grows and the indurated area enlarges. A genuine tumor develops, in the fundus of which there are often yellowish-red nodules the size of a pinhead on a red base—the carcinoma nests. Gradually the margins become prominent, rampart-like, and ever increasing parts of the lip become involved. This causes stiffness and immobility of the lip—symptoms which are often the first to send the patient to his physician. The tumor at this phase may undergo sudden advances, especially under inappropriate treatment with irritating ointments, etc.

Next there is swelling of the regional lymph glands, first of the submental, then of the sublingual. The cervical glands, at a greater distance, are next attacked. The symptoms increase largely if the tumor spreads to the gums and inferior maxilla. The lymph glands may attain to the size of large tumors. Suppuration and ichorization are constantly increasing, until the patient gradually succumbs to the disease. The possible duration of the disease has been calculated to be up to three and one-half years.

Treatment.—Radical operative removal of the tumor is imperative, as long as there is a chance of success. All attempts with

cautery or caustics are to be condemned. The excision should extend liberally into the healthy tissue. In very early operations permanent cures are frequent, and some statistics report up to 90 per cent. of successes. In inoperable cases, caustic pastes, radium and Roentgen-ray treatment may be tried, and the symptoms should meanwhile be relieved by the free use of narcotics.

Carcinoma of the Tongue.—Carcinoma often invades the tongue with signs of pronounced malignancy. This is due to the proximity of the buccal fundus, which is early involved, and in the loose tissues of which, abounding in lymph currents, the neoplasm spreads with great rapidity. Superficial carcinoma of the tongue, which distinctly preponderates, has histologically been proved to be typical carcinoma of the pavement epithelium, and is distinguished from deep, so-called glandular carcinoma of the tongue. The latter emanates from the deeper lingual layers and is at first located below the mucosa, without ulcerating, but later perforates the mucosa and ulcerates. Carcinoma of the cylindric epithelium of the tongue is very rare.

The affection begins as a small flat ulcer or a small nodular infiltration at the surface of the tongue, which is at first sharply demarcated from the surroundings. The border soon becomes less distinct, and the tumor penetrates from all sides into the lingual substance. Ulceration soon sets in, with a rampart-like thickening of the margins of the tumor. The appearance of the latter at this stage is particularly characteristic, greatly facilitating the diagnosis, especially in the presence of pain. Hemorrhages from mechanical causes occur easily. As long as the tumor is confined to the tongue the surrounding lymph glands remain unaffected, so far as palpation is able to establish. Should the growth extend to the inferior lingual surface and the buccal fundus, the tongue will become immovable, more and more interfering with mastication and deglutition. Stagnant food remnants cause repulsive ichorization which almost defies irrigation. Further development and metastases now proceed in the lymph glands, death finally ensuing from inanition. The average duration of the disease has been calculated at a little over a year. Everything, of course, depends upon an early diagnosis and an equally early operation. Both early detection and the necessary differential diagnosis may offer considerable difficulties. Experimental anti-syphilitic treatment should be carried out very energetically and very rapidly. A laboratory excision is likewise very useful. Differentiation between gumma and carcinoma offers the greatest difficulties, especially since both processes may be present, and because carcinoma may develop upon the soil of a gummatous ulcer. Metastases in internal organs are rare. For this reason, lingual carcinomata still belong to the comparatively benign forms

which offer good chances of a cure when operation is performed early.

Treatment.—The object of the treatment is the operative removal of the tumor. Statistics indicate that cures of more than three years' duration have been effected in about 30 per cent. of the cases treated by the present-day operative technic. This result is, of course, only possible when the involved glands can be simultaneously extirpated. The operation of today no longer encounters the difficulties of former times. In inoperable cases, narcotics and local anodyne measures are administered on a liberal scale. Ichorization can be minimized with iodoform gauze and irrigations. The lingual nerve is sometimes dissected for the purpose of alleviating the pain, which is oftentimes excruciating. In cases of this kind, treatment with the Roentgen ray and radium should be tried. The radium treatment is often successful in incipient cases.

Carcinoma of the Buccal Fundus.—Primary carcinoma of the buccal fundus is very rare. Superficial or deep carcinomata in this location produce functional disorders of the tongue at an early stage. The treatment is, of course, surgical, so far as possible.

Carcinoma of the Malar Mucosa.—Carcinoma of the malar mucosa is often found opposite carious teeth; most cases are superficial carcinoma of pavement epithelium. The seat of predilection is the hindmost part of the cheek pouches, whence the neoplasm easily extends to the intermaxillary fold, resulting in spasm in a short time. This may render the diagnosis very difficult. These carcinomata, however, do not run as malignant a course as those of the tongue, and are often locally restricted for a long period.

Treatment. Extirpation of the carcinoma, and plastic covering of the destroyed part of the malar mucosa. Lateral pharyngotomy, with resection of the submaxillary branch, may become necessary.

Carcinoma of the Palate.—Primary carcinoma of the palate is very rare. Its forms are: superficial, of pavement epithelium; and glandular, from the palatal glands. The superficial form is found most frequently in the posterior part of the hard palate, but also occasionally in the soft palate. As long as deep carcinoma is not ulcerating, it presents a flat tumor with a smooth hard surface which protrudes into the oral cavity. It may easily perforate into the nose, and can then be mistaken for gumma. The malignancy of palatal carcinoma is, generally speaking, less severe than that of carcinoma of the tongue or of the buccal fundus; the lymph glands, too, remain uninvolved for a long time.

Treatment.—The treatment depends upon the extent of the involvement. If the superior maxilla is involved, partial resection of that bone is the best remedy.

Carcinoma of the Uvula.—This affection is particularly rare, but often causes extensive metastases in the cervical lymph glands.

TUMORS OF THE MAXILLÆ.

Fibroma.—Fibromata may emanate from the periosteum of the alveolar processes and the inner wall of the alveoli. They often contain deposits of bone and lime, and are very hard. They are either pedunculated or broadly sessile, and may press the teeth before them out of the alveoli. As they grow larger they may bleed and ulcerate from mechanical contacts. There is another form of fibroma which starts from the true maxilla, by preference the inferior. It grows slowly, causing little disturbance, but may attain to an enormous size. Fibromata originating from the interior of the bone affect the inferior maxilla mostly. They may distend the bone considerably and perforate through the thin osseous shell. In doing so they may push the periosteum before them and cause neoplasms of bone, so that the entire tumor is covered with a thin osseous layer which crepitates upon pressure. Fibromata may change into cartilaginous or mucoid degeneration. Recurrences may follow operation, and these may at times be of a sarcomatous character. In the superior maxilla the fibromata may advance to the antrum of Highmore. The treatment can only be operative.

Chondroma.—Chondromata do not occur often at the maxillary bones, but may attain to an enormous size and destroy the bone.

Osteoma.—Osteomata may likewise occur at the maxillary bones. They fuse with the surrounding bone without forming any sharp border, or they may be encapsulated.

Odontoma.—Odontomata are tumors as hard as bone which have the exact appearance of dental tissue. The enormous enlargement of the lower maxilla which has been given the name of *leontias oris* is very rare. At times *exostoses* of the maxillary bones are met with.

Cysts.—Maxillary cysts are mostly due to disturbed dental development. The root cysts result from epithelial remnants which have been left behind in the development of the dental roots. These cysts often adhere to the apex of the dental root and may attain to considerable size. The cysts, like periostitic abscesses, may perforate toward the mouth, or less often toward the outer skin. Another form of cyst is developed through cystic degeneration of normal or supernumerary dental cells. It occurs in the maxillary body, in the nose or orbit, and contains dental fragments in various stages of development. Dermoid cysts are very rarely observed in the submaxillary bone.

Adamantoma.—These tumors are composed of enamel containing epithelium. They are usually located in the lower maxilla, the substance of which may be destroyed thereby to a considerable

extent. Although benign, their removal necessitates extensive maxillary resection.

Sarcoma.—The form best known is the sarcoma of the alveolar processes, sometimes called *epulis*—a giant-celled sarcoma which probably originates from the periosteum without forming metastases, but undergoes frequent recurrence. Unless operated upon early, it may attain to a considerable size, destroying large portions of the bone. The subjective symptoms are rather severe, because the tumor is injured by mastication and is then apt to ulcerate. Removal of the tumor by surgical intervention is easy.

Sarcoma of the Maxilla.—Sarcoma of the lower maxilla usually consists of spindle cells, that of the upper maxilla of round cells, the latter being distinguished for malignancy. Sarcomata of the lower maxilla often cause severe pain by exerting pressure upon the mandibular nerve. The object of the treatment is to remove these growths as early as possible.

Chondrosarcoma and Myxosarcoma run a malignant course, as a general rule.

Carcinoma.—Carcinoma of the pavement epithelium frequently develops at the alveolar processes, apparently from carious teeth or injuries to the gums. Central maxillary carcinoma occurs most frequently in the upper maxilla, the starting point usually being the mucosa of the antrum of Highmore. Central carcinoma of the lower maxilla is rarer. The treatment consists in resection of the affected bone.

AFFECTIONS OF THE PHARYNX.

Acute and Chronic Pharyngitis.—Acute catarrh of the pharynx occurs in infectious diseases and in acute inflammations of the mucous membrane of the upper air-passages; it may also be due to mechanical or chemical injuries to the pharyngeal mucosa. It subsides when the underlying cause is removed. No treatment is required except in aggravated cases, when irrigation and gargling of the pharynx with astringents and antiseptics (permanganate of potassium, tincture krameria, myrrh) are indicated. When the mucous membrane is much involved, gargling with warm chamomile or mallow tea or with a solution of cocain will have a pleasing effect. Lozenges containing anesthesin, orthoform, or cocain, slowly dissolved in the throat, are beneficial (see page 270).

Chronic catarrh of the pharynx is usually a manifestation of inflammatory processes of the nose and the nasopharyngeal space, in either the chronic or atrophic form, or in both combined, and usually affects the posterior and lateral parts of the pharynx. Excessive smoking, vitiated air, and sharp irritating food or beverages may likewise lead to chronic catarrh. The symptoms consist

of an unpleasant scratching sensation, accumulation of mucus, and slight pain in the pharynx.

Treatment.—Nasal catarrh demands attention. Smoking and the breathing of vitiated air are to be prohibited. Local application of 10-per-cent. silver nitrate or glycerin iodid (iodin 1, potassium iodid 1, glycerin 20) is indicated, as well as the use of suitable gargles and inhalants.

Follicular Tonsillitis.—Follicular tonsillitis is an infectious disease and runs a febrile course. It often begins with chills, and is characterized by ulcerous deposits in which the follicles are filled with caseous cores which often resemble diphtheritic or scarlatinal angina. In most cases both tonsils are affected.

Treatment.—When there is fever the patient is put to bed. Fluid diet, Priessnitz or ice compresses around the neck, and cold baths are indicated. Gargling with antiseptic fluids serves to clean and irrigate the tonsils. Severe headache may be relieved by acetylsalicylic acid and pyramidon. Some authors recommend inunction of the glandular region with Cr  d  's silver ointment (collargol). Between the attacks the tonsils should be enucleated.

Parenchymatous Tonsillitis, Suppurative Tonsillitis, or Quinsy.—This disease occurs as an acute affection, with the formation of a tonsillary abscess which is usually unilateral. It is associated with high fever and often with considerable disturbance of the general condition, severe pain, and greatly impeded deglutition.

Treatment.—The patient is put to bed, given small lumps of ice to swallow, and encouraged to gargle the throat. Ice compresses are applied. Morphin may be administered when the pains are very severe. Unless the abscess perforates spontaneously, it should be promptly incised; the tonsils are painted with cocain solution and an incision one or two centimeters deep is made, parallel to the teeth, toward the swelling. If, as may happen, the knife does not find the abscess, the latter will often perforate spontaneously without further assistance. After the pus has been evacuated the mouth should be frequently rinsed with boric acid solution, chamomile tea, or peroxid of hydrogen.

Chronic Tonsillitis.—Chronic tonsillitis may develop from the acute form, but it also occurs without any previous acute inflammation. It is characterized by the fact that it causes little complaint, but that nevertheless the tonsils persistently contain purulent and caseous cores in the crypts. These cores are often the source of rheumatic and septic affections, hence the importance of examining the tonsils carefully in cases of recurring articular rheumatism, endocarditis, and similar affections. In most of these cases, when the tonsils are affected they will have to be enucleated.

Hypertrophy of the Tonsils.—Many individuals, especially children, suffer from hypertrophy of the faucial tonsils. The enlargement may give rise to more or less discomfort, especially by

impeding the nasal inspiration and favoring the development of acute and chronic angina. It is advisable, therefore, to enucleate greatly enlarged tonsils. Pure air, sea air, saline baths, arsenic, iron and iodine often have a favorable effect upon the adenoid vegetations of the nasopharynx and hypertrophied tonsils, especially in children.

Retropharyngeal Abscess.—These abscesses are situated in front of the spinal column, and are attended with violent pains and, in advanced stages, with considerable dyspnea. They are the cause of the well-known typical position of the head, and can be palpated with the finger. They are exceedingly dangerous, as they may lead to edema of the glottis.

Treatment.—The abscess is opened early by a longitudinal incision, preferably without anesthesia, with the head hanging down to prevent the aspiration of pus. Large abscesses which are situated toward the side of the throat may be incised and drained from without.

Tuberculous Abscess.—Tuberculous gravitation abscess originates from necrosis of the vertebrae and may occur in the pharynx. The treatment consists in evacuation by incision from without and iodoform injections.

CHAPTER XVI.

DISEASES OF THE ESOPHAGUS.

ACCORDING to general acceptance, the esophagus is a musculo-membranous canal bounded superiorly by the cricoid cartilage. This cartilage and the adjoining part of the esophagus are movable upward and downward, especially when the head is strongly flexed forward or backward. The lower end of the esophagus fuses with the cardia without any distinct demarcation. The physiologic position of the cardia is between the ninth and eleventh dorsal vertebrae. Thus it follows that the position of the lower end of the esophagus depends upon the position of the cardia, perhaps also that of the diaphragm, which in the new-born lies comparatively high and in older persons lower.

The esophagus is divided into a cervical, a thoracic and an abdominal section; it is opportune also to distinguish between the supra- and infra-bifurcated parts—the parts above and below the bifurcation of the trachea. Taking into consideration the movability and changing altitude of the proximal and distal ends of the esophagus, the average length of this organ may be taken at about 25 centimeters (8 inches) in the male, somewhat less in the female. The distance from the upper incisor teeth to the cricoid cartilage is 15 centimeters, from the teeth to the cardia about 40 centimeters. The cervical section of the esophagus measures about 5 centimeters, the thoracic 18 centimeters, and the abdominal 2 centimeters. The distance from the teeth to the crossing point between esophagus and left bronchus is 23 centimeters. The supra-bifurcated part of the esophagus is from 10 to 10.5 centimeters in length.

The upper end of the esophageal lumen is closed by a sphincter, and opens but slightly even under exaggerated inspiration. This sphincteric demarcation between the pharynx and the esophagus is called the *os* of the esophagus. The esophageal walls approach each other in the cervical section, while in the infrabifurcated section there is an open lumen. Owing to compression of the muscle fibers of the diaphragm around the esophagus, the lumen is changed where the diaphragm is traversed. This change consists either in a diagonal fissure—right posterior to left anterior—or in a stellate opening. The cardia, when at rest, shows moderate tonic contraction and is closed. The esophageal lumen is not of uniform width. It is narrower at the cricoid cartilage, the aorta, the bifurcation, and the diaphragm.

The esophagus is a rather thick-walled muscular tube, invested with a thick, coarse layer of pavement epithelium. It is rather poor in glands and lymph follicles, in contradistinction to the complicated structure of the gastro-intestinal canal.

Deglutition Sounds. The two deglutition sounds important to the clinician were described by Meltzer thirty years ago. They can be distinctly heard with a stethoscope to the left of the xiphoid process. The first, or squirting sound, can be heard at the very beginning of the act of deglutition. It is a short, loud sound and indicates the absence of the tonus of the cardia. The second, or pressing sound, can be heard in normal cases six or seven seconds after the first sound. It indicates the passage of the swallowed fluid through the cardia when the latter is in a normal state of tonus; it is loud and long. These sounds assist in determining the permeability of the esophagus. In stricture or obstruction the second sound is delayed or absent.

INSTRUMENTAL EXAMINATION.

Esophageal Bougie. The esophageal bougie is of great value in the diagnosis of diseases of the esophagus, revealing the existence of obstruction due to a pocket, narrowing of the tube, or the presence of a foreign body. Before using the stiff sound, the much safer soft-rubber stomach tube should be employed. Should there be an obstruction, its character can be best ascertained by exploring the esophagus with the olive-pointed esophageal bougie, a flexible whalebone shaft with a screw at its distal end to which a metal

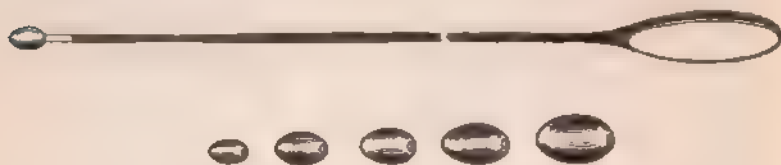


FIG. 65.—Olive-pointed esophageal bougie.

or ivory bougie of suitable size is attached (Fig. 65). The largest size is used first. With the instrument in the right hand, the olivary end is guided carefully back into the esophagus. When resistance, other than at the cricoid cartilage, is encountered, the shaft is to be marked at the incisor teeth with the finger of the left hand, for locating the obstruction on withdrawal. Smaller olive points are then employed until one is found which will pass the obstruction. Much force should never be exerted, as there is always danger of perforation. In case of obstruction at the cricoid cartilage the patient should be asked to swallow, and during the

act the bougie will easily slip through the esophagus into the stomach.

Esophagoscope and Gastroscope.—Since Mikulicz in 1881 employed a long straight metal tube with a light at the distal end to explore the esophagus and stomach, great progress has been made in both esophagoscopy and gastroscopy. Many instruments have been devised by Killian, Rosenheim, Kraus, Gottstein, Kuttner, Kelling and Gluecksman abroad, and Einhorn, Jackson, Plummer, Lerehe and Janeway in this country. All have added modifications, but the principle of the esophagoscope has remained the same.

The great value of the esophagoscope lies in the fact that with its aid the physician is able to see with the naked eye all parts of the esophagus down to the stomach. Not only has this method of direct visual inspection assisted greatly in diagnosis, but it is of material aid in treatment, as in the use of special forceps for the removal of foreign bodies, the application of drugs to ulcerated surfaces, curettes for the removal of sections for microscopic examination, and lens attachments for photographing pathologic lesions.

The best esophagoscope is that of Killian, with Bruning's handle lamp. Recently William Hill, of London, has modified Killian's instrument, making an esophago-gastroscope by adding a plane glass window at the proximal end, together with a faucet for attaching a rubber bulb for purposes of inflation, as in the case of the pneumatic sigmoidoscope (Figs. 66 and 67). By means of this one instrument the esophagus is examined by direct vision, and examination of the stomach by indirect vision is materially assisted (by inflation). The Hill instrument is a straight, rigid tube which can be easily passed under direct vision through the esophagus into the stomach. When the stomach is to be examined, an optical tube is inserted through the esophagoscope and the stomach is inflated with air.

Care and a working knowledge of the anatomy and pathology of the parts with which the esophagoscope must come in contact are, of course, necessary in the use of this instrument, the expert manipulation of which can only be acquired by experience.

When the esophagoscope is to be introduced, the patient, sitting on a low stool, with someone in a chair beside him supporting his back, throws his chest somewhat forward and his head as far back as possible. By this means the mouth, pharynx and esophagus are brought into line. The pharynx is thoroughly anesthetized with a 10-per-cent. solution of cocaine; the patient is instructed not to swallow immediately after the applications are made. The esophageal bougie, extending about six inches beyond the end of the esophagoscope, is introduced with it, the patient's head being dropped forward. As soon as the tip of the bougie passes over the epiglottis, the patient is instructed to throw his head back as far as possible, and the instruments are slowly thrust downward

until the end of the esophagoscope is felt to pass over the cricoid cartilage. This sensation is a very definite one to the operator. The bougie is then slowly removed, while the esophagoscope is held in position. By means of the Bruning handle lamp the light is then thrown into the esophagoscope, the further progress of which is under the constant guidance of the eye. In this manner there is practically no danger from the introduction.

Before inspecting the stomach, unless no food has been taken for ten hours previously, it is necessary to resort to lavage.

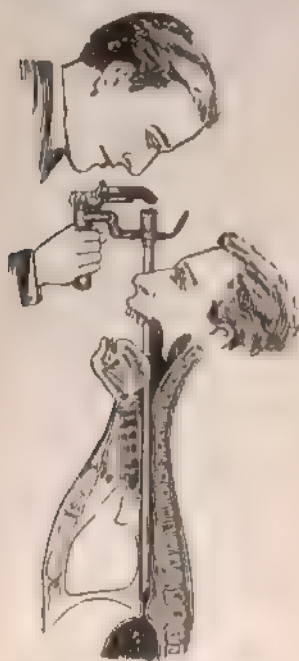


FIG. 66. Esophagoscope. (Hill.)



FIG. 67.—Gastroscope. (Hill.)

When passing the inner optical tube through the esophageal tube, inflation should begin before the end of the inner tube reaches the stomach, to prevent soiling the lamp and window of the periscope, which would obscure the vision. The whole mucous membrane of the stomach, including the pylorus, can be easily inspected. With a fully inflated stomach the rugae and convolutions of the mucosa almost entirely disappear, except in cases of chronic (alcoholic) gastritis. Engorged veins form a prominent feature in portal obstruction. Malignant growths, multiple hemorrhagic erosions, definite chronic peptic ulcers, hour-glass contractions and pyloric stenosis can be readily recognized. Rigidity and limited thickening of the stomach wall is suggestive of beginning carcinoma.

INFLAMMATION OF THE ESOPHAGUS.

Acute Esophagitis.—Simple acute catarrhal inflammation of the esophagus is caused by mechanical, chemical and thermic irritations. In rare cases catarrh of the pharynx and stomach may spread to the esophagus, and in equally rare cases acute bronchitis and laryngitis may be complicated by esophagitis. Sometimes the latter is associated with acute infectious diseases (measles, scarlet fever, typhoid, variola). In acute catarrh there is more or less pronounced hyperemia of the mucous membrane, with loosening and desquamation of the clouded epithelium. Occasionally the swollen glandlets of the mucosa protrude in the form of follicles which may lead to slight superficial ulceration (esophagitis follicularis).

Symptoms.—Probably acute esophagitis occurs oftener than it is diagnosed, for the reason that the symptoms are obscured or the neighboring organs are involved, enshrouding the esophageal affection. In pronounced esophagitis there are distinct piercing pains which may assume an intensely pressing and burning character, interfering with or entirely preventing the swallowing of food, particularly solid food. Even when the patient is at rest there is a dull pain deep in the chest, behind the sternum. There is often considerable thirst, less often moderate fever. A high degree of inflammation is often accompanied by a slimy, tough secretion, and in serious cases the food is brought up at once after entering the esophagus. The affection may also be associated with serious general nervous manifestations and a sensation of considerable angina. Patients are anxious to avoid any movements of the head. Pressure upon the sternum, palpation of the dorsal vertebrae, and movements of the body may be extremely painful. Introduction of the sound into the esophagus is always very painful in these cases and may cause spasm of that organ. Viewed with the esophagoscope, the mucous membrane appears swollen and relaxed, the vessels being injected.

Treatment.—If the affection can be traced to a definite cause, the latter must be treated. For the inflammation and thirst, iced milk and small pieces of ice are administered. In grave cases food should not be given by mouth, rectal alimentation being resorted to; gradually liquid and semisolid foods are allowed, until by degrees normal diet is resumed. Hot cataplasms are applied to the neck, chest, and along the spine; dry heat applied by hot-water bottles or thermophores often renders good service. Mustard dough sinapisms or capsicum plasters are applied between the shoulder-blades. Small doses of morphin are injected subcutaneously to relieve pain and reassure the patient.

Chronic Esophagitis.—Chronic esophagitis develops but rarely from the acute form. If, however, the irritation which causes

acute esophagitis repeats itself often, it may lead to the chronic form, or to chronic pharyngitis and chronic gastritis. An important rôle is played by the misuse of alcohol and tobacco, so that the affection is most frequently found in elderly men. It may also be due to persistent venous stasis in cardiac and pulmonary diseases, or it may occur as a complication with other esophageal affections, particularly in stenosis when food remnants become stagnant for a long time. Chronic affections of the stomach, in which acid and fermenting masses are often vomited, lead to chronic catarrh of the esophagus.

Symptoms. Pathologically, there are venous hyperemia, uneven thickening of the epithelium, and considerable secretion of mucus. When the affection has persisted for several years, there may be considerable hyperplasia of the mucous and muscular tissue. Polypoid and papillary proliferations of the mucous membrane are not rare. In other cases the affection causes but slight symptoms. In pronounced cases pain and pressure attend the act of swallowing. The principal symptoms are referable to the upper third of the esophagus, the walls here being in contact with each other. Often,

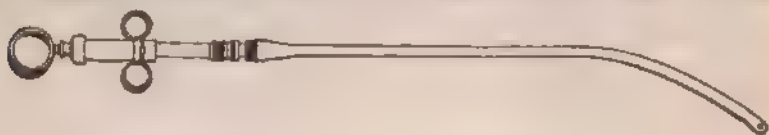


FIG. 68.—Esophageal syringe. (Rosenheim)

when there is great sensitiveness, the food is brought up, perhaps with an admixture of blood. Liquid food can always be better swallowed than solid. To establish the diagnosis, it is necessary to resort to the bougie and esophagoscope. The mucous membrane, as seen through the latter, is of a whitish cloudiness, or swollen and dark red, covered with a tough mucous secretion. The bougie is introduced to ascertain whether or not there is stricture.

Treatment. The use of tobacco and alcohol must be restricted. When the symptoms are quite severe the food should be soft, pappy and bland, and of medium temperature. The ingestion of good olive oil or sweet almond oil before partaking of food will facilitate the passage of the latter. Patients should beware of contracting colds. Skin irritation applied to the sternum and spinal column (sinapisms, tincture of iodine), or hot foot-baths, have sometimes a favorable effect. Local treatment is usually superfluous. When the pain is considerable, cocain 0.01 Gm. ($\frac{1}{8}$ grain), anesthesin or orthoform tablets may be taken; or equivalent medication may be introduced by means of Rosenheim's esophageal syringe (Fig. 68). This syringe holds 1 to 1.5 Cc. (15 to 25 minims) of fluid, and is provided with a long hard-rubber tube

which may be bent when heated. Cocain solutions (3 to 10 per cent.), eucain (3 to 5 per cent.) or silver nitrate (1 to 3 per cent.) may be injected into the esophagus, the syringe being long enough to reach the cardia. Another method of introducing antiphlogistic medication into the esophagus has been devised by Rosenheim; a soft-rubber stomach tube is covered with a paste that is rigid when cold, but is dissolved by the body heat. Such pastes are the following:

	Gm. or Cc	
R—Acidi tannici	0 15-0 5	gr. ii)-vii)
Olei theobromatis	10 0	℥iiss
Misce.		
R—Argenti nitratis	0 2 -0'5	gr. iv-viii)
Olei theobromatis	10 0	℥iiss
Misce.		

The tube is introduced down to the cardia and allowed to remain in position for about five minutes, by which time the paste has dissolved and acts immediately upon the mucous membrane.

Exfoliative Esophagitis.—This affection is an extreme exaggeration of acute esophagitis, inasmuch as the tendency to desquamation of the epithelium assumes such an extreme degree that large flakes of membrane become detached; these are often expelled in tubular pieces. An exudate forms beneath the injured epithelium, the latter being raised and detached as soon as there is sufficient extraneous cause. Before the membrane is definitely expelled there may be a temporary stenosis of the lumen accompanied by considerable dysphagia. The diagnosis of this affection cannot be made until the membrane has desquamated.

Treatment.—The treatment is the same as that of acute esophagitis. The use of the bougie is not advisable, owing to the danger of perforation or tearing off pieces of epithelium.

Fibrinous Esophagitis.—Necrotic fibrinous inflammation of the esophageal mucosa develops secondarily in grave general affections (sepsis, variola, cholera, typhoid, scarlatina, measles, tuberculosis, intestinal diseases of children). The clinical manifestations of this affection are usually so veiled by the general affection as to be completely overlooked, especially if there is a simultaneous affection of the tonsils, pharynx or larynx. Hemorrhages from the esophagus and expulsion of membranous shreds may point to the nature of the disease. The fibrinous inflammation may lead to extensive ulceration, and consequently to gradual constriction of the esophagus after the affection has run its course. The prognosis is always serious.

Treatment.—The treatment is directed toward the primary disease. For the local lesion it is symptomatic and expectant, just like that of simple esophagitis. The use of bougies or sounds is not permitted under any circumstances.

Phlegmonous Esophagitis. This disease, which is a rare one, may assume either the circumscribed or the diffuse form. It may originate from simple acute or follicular esophagitis, diphtheritic esophagitis, injuries inflicted by sounds or foreign bodies, or the spreading of phlegmonous processes in the stomach and pharynx; or from without by perforation of peri-esophageal pus foci, glandular abscesses, perichondritis, caseous lymph glands, or vertebral pathologic processes, into the external layers of the esophagus. In these latter cases the process diffuses in the submucosa and perforates through the mucosa into the esophageal lumen.

Phlegmonous esophagitis begins as a suppurative inflammation of the submucosa, with destruction of the tissue, and circumscribed or very extensive collections of pus under the mucous membrane. The undermined mucosa may then be pushed into the lumen of the esophagus to such an extent as to cause a constriction. Should the pus perforate into the esophageal lumen, this may lead to healing. It may also lead to fistular defects resembling diverticula, which, however, will not cause any particular disturbance. Perforation through the muscularis of the esophagus into the mediastinum or the thoracic cavities does not easily occur.

Aside from grave injury to the general condition, there are fever, dysphagia, and occasionally chill, in many cases nausea and dyspnea. The circumscribed phlegmons are prognostically favorable.

Treatment.—The treatment may at first be purely symptomatic. Ice and iced milk should be freely prescribed; rectal feeding may have to be resorted to. Morphin is often indispensable. If there is reason to suspect a phlegmonous affection, the bougie may be carefully introduced in order to forcibly open any possible abscess and evacuate the pus. This is a desirable proceeding, since spontaneous rupture is often considerably delayed, the patients becoming in the meantime much debilitated. If at all possible these cases should be examined with the esophagoscope, the abscess opened under guidance of the eye, and any foreign body removed as the cause of the trouble.

INFECTIOUS DISEASES IN THE ESOPHAGUS.

Diphtheria. Diphtheria of the pharynx and larynx may in rare cases spread to the esophagus, although this organ possesses such a degree of immunity toward diphtheria that the disease may spread to the stomach without affecting it at all. Diphtheria of the esophagus may easily be overlooked in the presence of diphtheria of the larynx. The symptoms are the same as in non-diphtheritic inflammation of the esophagus.

Treatment.—Antidiphtheritic and symptomatic.

Variola. -In variola an exanthem often develops on the esophageal mucosa in the form of hyperemic, infiltrated, granular efflorescences which may become superficially ulcerative. These changes will disappear with the disappearance of the original affection.

SKIN DISEASES IN THE ESOPHAGUS.

Pemphigus and *herpes zoster* have been observed in the esophagus by means of the esophagoscope. The treatment consists in non-irritating liquid diet, injections of cocaine or eucain, and administration of morphia.

BURNS AND CORROSIONS OF THE ESOPHAGUS.

Burns of the esophagus may occur through steam or hot liquids or solids. Esophageal burns due to steam are caused mostly by boiler explosions. Scalding and burning of the esophagus by hot liquids causes considerable swelling and reddening of the mucous membrane, which may become detached in shreds. The areas denuded of epithelium may slough, erode or ulcerate. Another possible sequel is constriction of the lumen due to scar formation.

Corrosion of the esophagus occurs through acids, notably sulphuric acid, and caustic alkalis, which are swallowed with the intention of poisoning or by accident. In light cases the epithelium is destroyed and desquamated - an injury which is easily remedied. In serious cases the corroded mucosa becomes acutely inflamed and necrotic; the necrotic parts are desquamated, and there is intense ulceration which may be followed by a phlegmon of the esophagus. When the ulcers gradually heal from the margin, the final result will be extensive scar formation and constriction of the esophagus. In grave cases the esophageal wall becomes necrotic in all its layers and is changed to a pappy tissue, leading invariably to death. The physiologically narrow regions of the esophagus are injured most by the corrosive fluid.

Immediately upon swallowing a corrosive substance, a violent burning pain under the sternum is experienced, followed by retching of pappy, hemorrhagic masses. Thirst, fever and collapse ensue. Unless the condition proves rapidly fatal, the pain gradually diminishes after twenty-four hours. After a time deglutition becomes less painful, and the injury may undergo healing. It may be possible for a patient to swallow small pieces of solid food; but as soon as scars begin to form, the lumen is constricted and swallowing is again interfered with. This undesirable result cannot be avoided except in light cases.

Statistics have shown that more than half the cases of sulphuric acid poisoning terminate fatally, and about 25 per cent. of alkali poisoning cases. In more than one-third of the survivors of sul-

phuric acid poisoning, and more than one-half of those who survive alkali poisoning, grave constrictions result. About one-third of the strictured cases die as a consequence of the strictures.

Treatment.—In acid poisoning, magnesia is administered by the teaspoonful in iced milk. In alkali poisoning, diluted acetic or citric acid is given in iced water. These remedies must, of course, be given immediately after the poisoning, to have a neutralizing effect. Otherwise there is nothing but symptomatic treatment to overcome the inflammation, such as the swallowing of small pieces of ice, application of ice-bags to the chest and neck, narcotics, rectal feeding, and restriction of mouth feeding to liquid foods. Solid food is not permitted for a long time, even in favorable cases. When there is considerable dysphagia after the acute manifestations have subsided, the chest and the spine are to be painted with tincture of iodine; or a mustard poultice may be applied. No instrument should be introduced in fresh cases of corrosive esophagitis, as long as necrosis or fresh ulcerations are present, which often means for three or four weeks. Epinephrin, 5 to 10 drops of the 1:1000 solution in a teaspoonful of water, gives great relief. The stricture is to be treated later (see page 365).

It is not necessary to resort to gastrostomy in each case of fresh corrosion, although some authors advise it. This operation becomes necessary when the injury runs a chronic course and when large pieces of the esophagus are expelled, with the probability of deep ulcerations and extensive cicatrization. Gastrostomy is also necessary if, at an early stage, acute swelling causes complete occlusion of the esophagus, or when the formation of peri-esophageal abscesses is suspected. In these cases the stomach fistula is necessary, that the strength of the patient may be maintained and the esophagus spared.

ULCERS OF THE ESOPHAGUS.

Gangrenous Ulcers.—Gangrenous processes in the neighborhood of the esophagus (pulmonary gangrene, noma, gangrene of the tonsils) may spread to the esophagus and cause grave destruction, but this is a rare occurrence.

Gangrenous ulcers may also be due to pressure upon the esophagus from within or without. Impacted foreign bodies may render the mucous membrane and the deeper layers necrotic by persistent pressure; permanent sounds may exert a similar effect; ulcers or aneurysms may cause circumscribed gangrene by pressure upon the esophageal wall from without.

Decubital Ulcers.—Decubital ulcers are caused in the esophagus in the same manner as in any other part of the body when, aside from any local circulatory impediment, there is a tendency to the formation of such ulcers. Such a tendency may be due to old

age, general affections (infectious diseases), or neurotic affections. Generally speaking, there is only one point in the esophagus, or rather in the lower part of the pharynx, which inclines to the formation of these ulcers, and that is at the cricoid cartilage. In patients suffering from a serious disease, or who are bed-ridden, as well as in cases of senility, the broad, thick part of the cricoid cartilage lies directly upon the posterior pharyngeal wall, especially when the body is in the horizontal position. This persistent pressure causes a decubital ulcer of the mucosa just at the point where it covers the cricoid cartilage, and another one precisely opposite on the mucosa of the posterior pharyngeal wall—the latter being, to a certain extent, a counter-product of the ulcer of the cricoid cartilage. These ulcers occur only in the last few days or weeks of life, and therefore do not involve the question of treatment.

Tuberculous Ulcers.—Esophageal tuberculosis occurs as a concomitant manifestation of grave tuberculosis in other organs. Usually there are superficial ulcers of various sizes, with either a smooth or a rough base. On this ulcerous base, at the margins, or in the neighborhood of the ulcers, characteristic tubercles can be seen underneath the epithelium which as yet is intact. The affection is probably due in most cases to perforation of caseous lymph glands into the esophagus. It certainly is possible, by way of exception, for tuberculous sputum to cause the infection, especially when the mucosa is already injured from other causes. The disease may run its course without causing any symptoms, but, on the other hand, it may give rise to pains and dysphagia. There is also a possibility of esophageal stricture developing in the course of the disease. The *diagnosis* can be made with the esophagoscope (see page 352, Figs. 66 and 67). The *prognosis*, as a matter of course, is unfavorable.

Treatment.—Treatment is purely symptomatic and consists in injections of a 3- to 10-per-cent. cocain solution with the esophageal syringe (Fig. 68) prior to the ingestion of food; or the administration of oil, or of anesthesin, orthoform, or bismuth subnitrate in water, before eating. The food should be liquid, pappy and non-irritating.

Syphilitic Ulcers.—It is an established fact that, in the tertiary period of acquired syphilis, submucous gummata and diffuse ulcerating syphilitic infiltrations occur, in rare cases, in the esophagus. The gummata may either ulcerate or shrink to fibers. Congenital syphilis, too, appears to be capable of causing esophageal infection. It is not yet certain whether the esophageal mucosa may be affected in the secondary stage. The symptoms are dysphagia and painful deglutition. When the infiltrates and ulcers heal, extensive scars and strictures may remain.

Diagnosis.—The diagnosis is based upon the presence of other syphilitic symptoms, upon the results of antisyphilitic treatment, or, occasionally, upon esophagoscopic observations. The Wassermann test assists in the diagnosis.

Treatment.—The treatment is antisyphilitic—iodids, mercury, and arsphenamine (see Chapter XXVIII). If there is any tendency to stricture, the use of the sound is indicated.

Peptic Ulcer.—As a matter of fact, ulcers may occur in the esophagus which entirely correspond to the round ulcers of the stomach. The seat of these peptic ulcers is in most cases the lower third of the esophagus. They are caused by the acid gastric juice, and occur principally in persons with hyperacidity, as a consequence of frequent eructations with regurgitation of the acid gastric contents, or from the escape of the acid gastric juice from the stomach into the esophagus due to defective closure of the cardia. They may be single or multiple, or they may occur simultaneously with gastric and duodenal ulcers. Peptic esophageal ulcers are round, like gastric ulcers, of varying sizes, have smooth edges, contain brownish-black tissue shreds at the base, and, like gastric ulcers, have a tendency to grow into the deep parts. Such an ulceration may be exceedingly large, or it may grow around the esophagus circularly, giving rise to pouches and valves, simulating stenosis. As a rule the ulcers tend to heal by cicatrization. Small ulcers heal without any particular sequelae, while large ones may result in stricture. Very deep ulcers may easily cause adhesions with surrounding parts and perforation into the mediastinum, pleura, etc. As a matter of course such an ulcer may degenerate into a carcinoma.

Symptoms.—The chief symptom of the ulcer is pain in swallowing, which is usually felt at the level of the xiphoid cartilage. Solid food usually gives greater pain than liquid. There may also be pain after eating. Sounding of the esophagus causes pain at the seat of the ulcer. Sometimes the stomach is the seat of severe pain. There is often a tendency to pyrosis, sour eructation, and vomiting. Hematemesis and hemorrhagic stools may occur, as in gastric ulcer. The ingestion of food may become so difficult that the patients lose flesh rapidly. Under certain circumstances the ulcer is visible with the esophagoscope; otherwise the diagnosis is always difficult.

Treatment.—It is advisable for the patient to forego eating for a number of days, depending entirely upon rectal feeding. This should be followed by ulcer treatment according to Leube or Lenhartz (see Chapter XXV). This treatment is carried out as in gastric ulcer, and includes absolute rest in bed. Hyperacidity is energetically counteracted by mineral waters, the administration of alkalis, and liberal doses of belladonna—0.01 to 0.03 Gm. ($\frac{1}{8}$ to $\frac{1}{2}$ grain). Pain is relieved by cocain tablets 0.01 Gm. ($\frac{1}{8}$ grain),

anesthesin in powder or tablets 0.03 Gm. ($\frac{1}{2}$ grain), or orthoform 0.03 Gm. ($\frac{1}{2}$ grain), or one of the last two in conjunction with bismuth. In grave cases, when other measures fail and the patient is losing ground through lack of food, a gastrostomy is required. When, after healing of the ulcer, there are symptoms of stricture, the sound must be regularly used (see page 365).

VEGETABLE AND ANIMAL PARASITES.

Actinomycosis.—Primary actinomycosis of the esophagus is very rare. The diagnosis is made with the esophagoscope, and is, of course, comparatively easy when infected material can be removed from the ulcer.

Thrush.—Thrush of the esophagus occurs comparatively often. It is the result of direct spreading of a thrush infection of the mouth and fauces (see page 309) to the esophagus, where it grows exactly as on the oral mucosa, so that the mycelia often extend through the mucous membrane and into the muscle. The infected areas appear on the esophageal mucosa either as small flat or lumpy deposits, or as striated irregular plaques and membranes; or else the vegetation is so extensive that the esophagus is lined with it for long distances or throughout its entire length, or it may be filled with dense, compact thrush masses to such an extent that occasionally tubular casts of the esophagus are expelled. The mucosa underneath the proliferations is inflamed. After the plaques have desquamated, a flat loss of substance remains.

Esophageal thrush causes no symptoms except when markedly developed, and it may be impossible to make the diagnosis during the patient's lifetime. Swallowing is apt to become difficult or impossible, especially in children. Since extensive development occurs only in the presence of grave general infections and in diabetics, the question of treatment need hardly be considered.

Animal Parasites.—*Leeches, flies, ascarides* and *wasps* have been found in the esophagus. In one case a wasp sting caused serious esophagitis. Occasionally, the transversely striated musculature of the esophagus contains free or encapsulated *trichina* when general trichinosis is present (see Chapter LII).

STRICTURE OF THE ESOPHAGUS.

The various forms of esophageal stricture may be classified as follows:

1. Stricture due to an affection of the esophageal wall (neoplasms; cicatricial strictures following corrosions and ulcerous processes; diverticula).
2. Stricture due to occlusion of the lumen (foreign bodies, thrush).
3. Stricture due to compression of the esophagus from without (affections of the neighboring organs).

4. (a) Spasm of the esophagus; (b) Dilatation of the esophagus.
5. Congenital stricture.

Neoplasms in the Esophagus.—Papilloma.—Papillomata in the esophagus possess merely an anatomic interest, as they cause no symptoms whatever. They are from pinhead to pea size, grow on the surface of the mucosa, and originate from elongation of the papillæ of the mucosa and thickening of their epithelial covering. Anatomically they correspond to the ordinary warts of the skin, and are, for the most part, found in old people, either isolated or in large numbers, and often accompanied by carcinoma.

Fibroma.—Fibromata are the most frequent of benign neoplasms of the esophagus. They start from the submucosa and muscularis and become more or less prominent tumors, of soft consistency and an uneven lobular surface. These fibromata never cause complete occlusion of the esophagus, owing to their softness and the fact that the opposite part of the esophageal wall evades the tumor, so that most of the food can pass unhindered. Fibromata may easily cause symptoms if they grow in pedunculated form on the surface and represent polypi, as is often the case. They occasionally attain to such a considerable size that they are sure to cause disturbance, such as: painful pressure in the chest, increased during meals; pressure on the trachea, interfering with breathing; or dysphagia independent of this symptom. The polypi are frequently located in the upper third of the esophagus, and the act of vomiting or retching may thrust them upward, causing pressure upon the epiglottis and threatening suffocation. The polypi may even become palpable and visible in the pharynx and mouth—a fact of diagnostic importance. Sometimes inspection through the esophagoscope renders the diagnosis clear; or it may be necessary to excise a small piece of the tumor and examine it microscopically. These benign polypi may cause certain dangers, owing to their location, necessitating their removal. The treatment is entirely surgical.

Other Benign Neoplasms.—*Lipomata* have been observed in the shape of sharply demarcated spheroidal tumors or polypi; also pedunculated or unpedunculated *myomata*, the former causing manifestations similar to those of fibrous polypi. Idiopathic *hypertrophy* of the esophageal musculature has likewise been observed. The mucous glands sometimes develop into *cysts*. All these kinds of tumor hardly cause any clinical manifestations. *Dermoids*, which in most cases occur in the lowest part of the pharynx, are exceedingly rare.

Carcinoma.—Among tumors of the esophagus, carcinoma is the most frequent. In the great majority of cases it consists of pavement epithelium. The tumor originates either from the deep epithelial layers of the mucous membrane or from the epithelium of the excretory ducts of the mucous glands. The process of

extension is more often annular than in the form of separated areas of erosion. The surface of the carcinoma is nearly always ulcerated, and the surrounding mucous membrane is in most cases chronically inflamed. The esophagus is never affected in its entire length. Points of predilection for the development of carcinoma are the region of the bifurcation of the trachea, and that part of the esophagus which is surrounded by the diaphragm (*hiatus oesophageus*). Most esophageal carcinomata are located at the level of the bifurcation of the trachea. Predisposing factors favoring the development of carcinoma are scars, excoriations, and esophageal ulcers. Further contributing causes are chronic mechanical, chemical or thermic irritations, stirring up the points of predilection above referred to, which represent the physiologic constrictions of the esophagus. For this reason, alcoholics are especially liable to the affection. The majority of cases are furnished by the male sex, mostly between the ages of forty and sixty.

If food lodges and is retained above the carcinoma, dilatation with hypertrophy of the musculature may develop; but this does not often happen, because in most cases the food is promptly vomited. Dilatation may also occur above the carcinomatous stricture, if any food passing through is not carried onward by peristalsis.

Symptoms.—The first and most important symptom is interference with deglutition. In most cases this disturbance develops quite gradually, commencing with slight pressure behind the sternum when solid morsels are swallowed. The difficulty increases more or less rapidly until nothing but thin liquids can be swallowed, and even these not without effort. Again, dysphagia may set in quite suddenly, or it may undergo rapid improvement owing to necrotic decay of large tumor masses. As the stricture increases, the food is often vomited together with mucus, saliva, and blood. The presence of much pain suggests further spreading of the carcinoma and metastatic formations which exert pressure upon neighboring nerve trunks. The sensation of hunger, often experienced, gives way to pronounced anorexia. In most cases there is much thirst. As the tumor grows, involving neighboring organs, there may be hoarseness, aphonia, dyspnea, and paroxysms of pain. It not infrequently happens that there is unilateral paralysis of the vocal cords which can only be recognized with the laryngoscope. Furthermore, the carcinoma may perforate into the trachea, mediastinum, or large vessels, fatal hemorrhage resulting.

Diagnosis.—The diagnosis is made from the anamnesis; from the sound findings which will establish the presence and seat of the stricture; from Roentgen-ray examinations (Plate XII, Figs. 1 and 2) which reveal its length; or through the esophagoscope revealing the ulcerating mucosa. Sometimes small particles of the tumor can be withdrawn with the tube or the fenestrated sound

for purposes of microscopic examination, or small parts may be excised in the esophagoscope. If stagnant food remnants can be withdrawn from above the stricture, they will be found free from hydrochloric acid, and the presence of blood can be microscopically demonstrated, also pus corpuscles, and often many long bacilli.

The affection usually terminates fatally, on the average after about ten months. Surgery alone can bring about complete relief. Recently radium has been found to be of some benefit, retarding the development of the neoplasm (see page 550).

Sarcoma.—Sarcoma as a primary affection is exceedingly rare, but as a secondary manifestation it may spread from neighboring sarcomatous organs. The symptoms resemble those of carcinoma. The diagnosis is assured by the esophagoscope and excision for the laboratory.

Cicatricial Stricture.—It has already been stated that cicatricial strictures occur after corrosion of the esophagus and cicatrization of ulcerous processes (tuberculosis, syphilis, peptic ulcer).

Treatment of Esophageal Stricture.—In the treatment of stricture of the esophagus, the use of the sound is of prime importance. When the tip of the olive-pointed bougie meets an obstruction, this is due either to a pocket or to narrowing of the lumen of the esophagus. Under such circumstances Mixer's method of using silk thread as a guide to the bougie should be adopted. The patient is allowed to swallow gradually six yards or more of strong silk thread. After the thread has passed through the different segments of the intestine, it cannot very easily be withdrawn through the mouth. Traction on the upper end makes the thread tense, and the olive-pointed sound is now passed on the thread (Fig. 72). When introduced on a taut thread, the sound follows the axis of the lumen of the esophagus. By varying the tension on the thread, obstruction from pocketing and obstruction from actual narrowing of the canal may be distinguished (Plummer). Figs. 69 and 70 illustrate the use of a sound in demonstrating the existence of a pocket. The sound is first introduced into the diverticulum until obstruction is encountered; holding the sound in place, the thread is drawn taut. Traction on the thread will now lift the sound out of the diverticulum sufficiently far to bring the point to a level with the opening into the esophagus. With the silk thread as a guide, greater force can be used than would otherwise be safe in dilating a stricture of the esophagus.

Solid sounds are useful, and those principally in use are of English make, measuring about 60 to 80 centimeters in length. They are made of tissue impregnated with resin, wax and lacquer; are flexible; have a perfectly smooth surface and either a cylindrical body rounded at the point or a conical shape tapering at the point. These sounds are manufactured in all diameters.

Before use, they are immersed in warm water or rubbed with a cloth, to render them more flexible. The correct degree of hardness is attained when the horizontally held instrument bends downward by its own weight. Recently sounds have been manufactured of duret. By a special method of treatment, great resistance is imparted to this rubber, which may also be boiled without injury.

Hollow sounds, as well as solid ones, are in use. They are made of tissue covered with resin, wax and lacquer, and are provided with one or two openings at the lower end. The object is to combine the sound examination with simultaneous artificial feeding.



FIG. 69. Olive-pointed sound and thread in diverticulum.

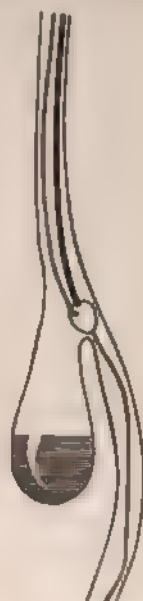


FIG. 70. Olive-pointed sound and thread guided into esophagus.

For this purpose the instrument is enlarged at the upper end to hold a perforated wooden cone, which communicates through a tube with a funnel or irrigator into which nourishing liquids may be poured. Instead of making use of a wooden cone, the funnel or irrigator may be connected with a wide rubber tube that can be pulled over the end of the sound.

Technic of Introducing the Sound.—The patient is informed of what is about to be done, that the procedure is painless, and that he may continue breathing regularly. This is important in order to prevent nervous retching and vomiting. The patient sits comfortably upon a chair, leaning back for support, with his head erect or bent very slightly backward. A rubber cloth or towel

is fastened at his neck, and he is instructed to hold a basin with both hands. He is also told not to swallow any sputum or ascending food remnants, but to empty them into the basin. Artificial teeth are removed before the procedure begins. The sound, having been immersed in warm water or lubricated with oil or glycerin, is held by the operator with his right hand, like a penholder. The first and second fingers of the left hand are inserted into the patient's mouth up to the posterior faucial wall, and the sound is introduced along the right side of the first finger or between the two fingers up to the faucial wall. At this point the cricoid cartilage, as a posterior eminence, offers resistance. To overcome this obstacle the patient is instructed to swallow several times, or the tongue is energetically depressed forward and downward from behind by means of the first or the first and second fingers of the left hand. This also serves to push the epiglottis forward, so that the cricoid cartilage is moved away from the vertebral column. The sound is now at liberty to enter the esophagus, where it glides down almost by itself. It may happen, however, that the sound slips into one of the pyriform sinuses, in which case it will refuse to go down in spite of all coaxing. It would be wrong to use force, the better plan being to withdraw the instrument and commence afresh. It may also happen that the sound finds its way into the larynx, an accident which is usually accompanied by considerable coughing and dyspnea, thus clearing up the situation immediately. The sound should then be withdrawn, and reintroduced after the patient has calmed himself. As soon as the sound lies correctly in the esophagus, so-called sound-breathing may be observed, which consists in a light motion of air through the sound, caused by the pressure vacillations within the thorax.

After the sound has passed through the esophageal entrance, slight pushing will cause it to glide down, passing the cardia without resistance, and it will be arrested only by the greater curvature. It is important to assure one's self that the upper incisors do not create any friction with the sound, as this would give the erroneous sensation of resistance in the esophagus.

If in this way the thickest sound or the stomach tube glides into the stomach without resistance, there can be no question of any obstacle in the esophagus. Should any obstacle be encountered, the operator, detecting its presence by the guiding hand, must determine its location. This is done by marking the sound *in situ* at the point where the incisors touch it, and, upon withdrawal, measuring the distance from this point to the end of the sound. (See schedule of distances, page 349.)

The location of the obstacle being determined, the question arises, how to enter the stricture for the purpose of overcoming it. The simplest way is to proceed gradually to the use of thinner and thinner sounds until one is found which will pass. The sensation

of the sound passing through is experienced by the operator's guiding hand. It is frequently possible to feel the approximate length of the stricture. There is often a sensation as if the sound were passing through a spiral canal, and this is found in many cases to be true.

The stricture may be so narrow that even the thinnest sound will not pass, or the opening may be so situated that the straight sound cannot find it. In such a case the use of force, if the stenosis is carcinomatous, may result in perforation; in cicatricial stricture a little additional pressure may be employed, but even then caution is necessary. It may happen that the sound passes freely one day but not on the following day; this is probably due to the irritation caused by the procedure, especially in carcinomatous stenosis, where an inflammation caused by the sound may still further constrict the lumen. It may then be opportune to leave the stricture undisturbed for a few days, resorting to rectal feeding if necessary. If, after the inflammatory manifestations have subsided, the sound cannot be introduced, a little artifice may help. By bending the lower end of the sound slightly, and, when inserted, gently rotating it, carefully withdrawing and reinserting, it is often possible to find a passage through an eccentrically situated

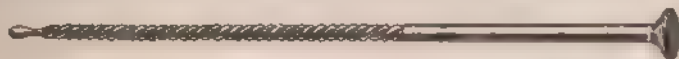


FIG. 71.—Esophageal sound. (Crawcour.)

stricture. It may also be desirable to change the cylindric sound for a conical one. Several thin, well-lubricated sounds may be introduced simultaneously in the hope of passing the stricture by advancing the various sounds alternately. When an ordinary English sound fails, entrance to the stricture may sometimes be effected with Crawcour's sound (Fig. 71). This is a straight button sound, made of steel, rigid at the top with a solid handle, but flexible in the lower section, which is a spiral of rolled plate metal. It is made in diameters of 6 to 10 millimeters. Sometimes these sounds pass the stricture without pressure, owing to their flexibility and weight. They can easily be kept clean and are then of great durability. They are cleansed with water or antiseptic fluid, such as lysol, and dried warm. Before use they are best moistened with water. Should even the use of this sound prove unsuccessful, the entrance to the stricture may perhaps be found by the esophagoscope, and the sound introduced under the direct guidance of the eye. For this purpose the ordinary English sounds are available. If these should fail, the thread and bougie should be used as described on page 369.

Should none of these methods be successful, no further attempts should be made at the time. If the patient cannot be fed by mouth,

nutritive enemata should be given; he should rest in bed and the sensation of hunger and thirst should be relieved by regular rinsing of the mouth with ice-water. Small doses of cocain, 0.01 to 0.02 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain), twice or three times daily, or small doses of morphin, may be administered. In this way it may happen that a previously impassable stricture becomes permeable. If not, and the patient continues to lose in weight, we must regard the case as one of practically impermeable stricture and recommend gastrostomy. On the other hand, it may be possible for the patient to swallow liquid food although a sound cannot be passed. Generally speaking, operation in these cases of carcinomatous stenosis should be deferred as long as possible, provided the emaciation is not extreme. The benefit to be derived from gastrostomy is, of course, not very great, life being prolonged, on the average, about five months. In cicatricial strictures, however, the operation should on no account be delayed until the patient has arrived at the last ebb of his strength, because after gastrostomy and the improvement of nutrition there is still a possibility of relieving the stricture either from above or through the stomach. In very debilitated carcinomatous patients it is preferable to provide an intestinal fistula by jejunostomy.

Dilating the Stricture.—The stricture being permeated by one of the sounds, the next task is to dilate it. This is of particular importance in cicatricial strictures, but should also be attempted in the carcinomatous form. When the location of the stricture is simple, the largest passing sound is allowed to remain *in situ* for some time, if possible for fifteen to thirty minutes, the patient being instructed to breathe quietly and to empty sputum and mucus into a basin. Unless the patient is excited or overstrained, the next larger sound may then be introduced and allowed to remain for some time; otherwise the procedure is deferred until the next day. The sound which has already passed is then introduced first, and upon withdrawal is followed immediately by the next higher number. According to how the patient bears the procedure, the applications may be made daily or every second day. In this way the size of the sound is constantly increased, be it the cylindric, conical or Crawcour variety. Great caution is always necessary in carcinomatous stenosis, while in cicatricial strictures more force may be applied, the largest sound being used in the course of time. In that case lubrication of the sound is preferable to moistening with water. Permanent good results have often been attained in cicatricial strictures by this progressive dilatation. Should the scar contract again, as it may, the treatment may have to be repeated. In esophageal carcinoma it would not be correct to force the sound treatment, especially when the passing of the instrument encounters difficulties. The injury to which the patient would be thus exposed is greater than the benefit from moderate

dilatation, and there is always the danger of hemorrhage and perforation to be reckoned with.

As soon as the stricture has been dilated to admit medium-sized sounds, the question of using hollow sounds and feeding artificially thereby has to be considered. In cases in which the very finest English sound cannot pass through, it may still be possible to effect a slight dilatation by introducing well-rounded, defibrinated gut strings, which would swell in the stricture. As these strings may be allowed to remain for several hours, the result may be at least a dilatation sufficient to admit the finest solid sound. The strings may be introduced through the esophagoscope under the guidance of the eye, several fine and coarser ones being pushed through in the hope that one or more of them may penetrate the stricture.

The Sippy Dilator. B. W. Sippy has perfected a system of gradual dilatation of esophageal stricture by the use of a series of conical metal bulbs strung on a piano wire concentrically—a small bulb at the extreme end, held in place by a still smaller bulb welded to the wire, and followed by three or four others, each larger than the one preceding it, and all pointed toward the end; then an equal number of bulbs pointing in the other direction and gradually decreasing in size, so that the appearance is that of a string of beads, the largest in the center. The piano wire is four feet long, No. 20, reduced in size for greater flexibility for a distance of eight inches from the end. Before the conical bulbs are strung upon it, the permeability of the stricture is determined as follows: The silk thread, introduced and anchored in the manner described on page 364, is threaded through the small metal bulb at the end of the piano wire, so that the thread, held taut, serves as a guide for the wire and its terminal bulb, as well as for the conical bulbs that are to be later strung upon it. The sounding is begun with one of the small dilators, or conical bulbs, which is held snugly against the terminal bulb by a spiral "introducer" twenty inches in length that is slipped on the wire from the free end. If the dilator passes down the esophagus freely, a larger size is chosen, and so on until a snug fit is found. This is removed and the wire is strung with the graded series of dilators or conical bulbs as already described, the largest being just a trifle larger than the bulb which has been found to fit the stricture. The spiral introducer, with a bulb attached that is a little smaller than the upper terminal bulb on the wire, is then slipped on the wire and pressed against the row of strung bulbs; the latter passes gradually down the esophagus until the central dilators have passed the stricture. The stricture, having been thus dilated from above, is dilated from below as the bulbs are drawn upward by traction on the free end of the wire. The pressure is almost entirely lateral, owing to the conical form of the dilating bulbs; and the flexibility of the whole apparatus, which is nevertheless held securely in the esophageal channel, is a most commendable feature. Sippy says

that by this method the most tortuous stricture may be dilated with the minimum degree of traumatism. While dilatation is proceeding, the silk thread by which the wire and its loop are guided is held by an assistant, for the operator requires both hands to conduct the

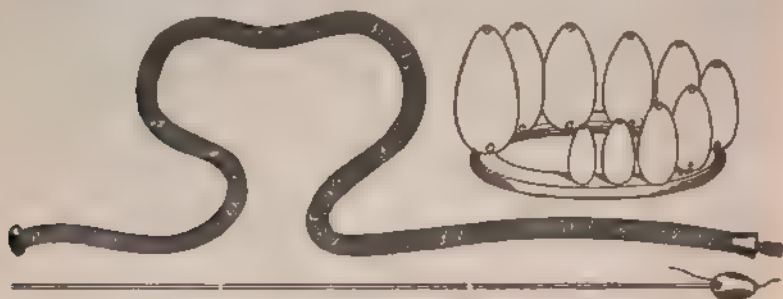


FIG. 72.—Esophageal dilator. (Sippy.)

advance and return of the dilators—one for the wire and the other for the introducer. The Sippy apparatus is shown in Fig. 72.

Senator devised a dilating instrument consisting of a soft French sound provided with a metal end, to which a thin laminaria tent



FIG. 73. Esophageal dilator. (Senator.)

of varying thicknesses can be connected by means of a screw. The tent is also secured by two silk threads (Fig. 73). The sound is introduced far enough to allow the tent to lie in the stricture. This may be done in the esophagoscope under guidance of the

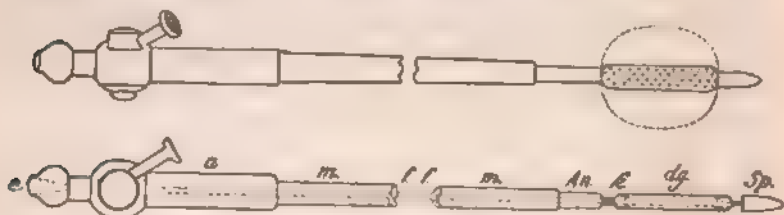


FIG. 74.—Esophageal dilator. (Schreiber.)

eye, the tent being first moistened with water so that it will swell when placed in position. The sound is then detached (unscrewed), and the tent, with its silk threads hanging out of the patient's mouth, is allowed to remain for several hours. The tent should

not remain in the stricture long enough to render its withdrawal difficult on account of increase in volume. When withdrawn it should be cleansed with water and dried, after which it can be used again. Some authors have achieved good results with this treatment.

Another means for energetically dilating a permeable stricture is the dilating sound of Schreiber (Fig. 74). This instrument is a metal sound with a small rubber balloon attachment. A thin whalebone, introduced into this sound from its lower end, can be screwed to the metal lengthening piece at *k*; it carries a conical end-piece (*Sp*) and the rubber balloon (*d.g.*). The upper and lower ends of this rubber balloon are fastened with silk threads. A syringe holding about 10 Cc. of water is provided with a conical mouth-piece which fits exactly into *e*. To remove the air in the sound, the piston of the syringe is withdrawn once or twice, the sound being held in an upright position. The syringe being removed, the little water contained in the rubber balloon is squeezed out by compression.

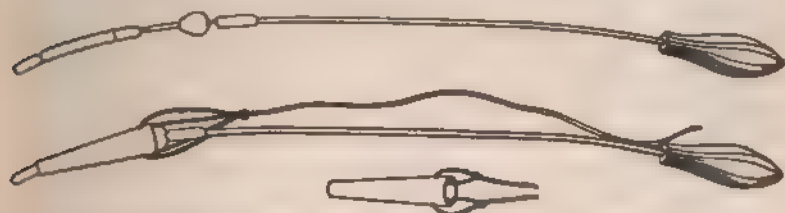


FIG. 75 — Esophageal bougie and cannula. (Leyden and Renvers.)

The cock is now closed and the sound introduced. In a normal esophagus the sound glides smoothly into the stomach. If now about 10 Cc. of water be injected into the rubber balloon, it fills out to a circumference of 7 to 9 centimeters, not too large to prevent it from passing a normal cardia upon withdrawal of the sound, but it is always arrested by the cricoid cartilage at the esophageal entrance, so that the cock has to be opened to permit of its withdrawal. If there is a stricture in the esophagus, the balloon is arrested sooner, so that the instrument is to a certain extent of diagnostic service. Besides, the balloon can be used for dilating the stricture, being either drawn, filled with water, into the stricture from below and allowed to remain there, or introduced empty by means of the sound into the stricture (provided the location of the latter is known) and then distended with water. With this instrument it is possible to exert uniform pressure, without any danger of injury.

Sounds for esophageal strictures have also been devised by Leyden and Renvers, with the object of inserting a cannula after the manner of laryngeal intubations, to keep the esophageal stric-

ture permeable and render the introduction of sufficient food possible. These permanent cannulae (Fig. 75) are 5 to 10 centimeters long, with an upper lumen of 12 to 14 millimeters and a lower of 5 to 6; but they are manufactured in different sizes. The upper end of the cannula is not round, but flattened from front to back, so that the cannula may pass the cricoid cartilage more easily as it is withdrawn. At both sides of the upper end of the cannula there are apertures for coarse silk threads which are fastened to the sound by a clamp at the time of insertion. The sound itself is a bougie with a handle, and is equipped with two ivory buttons, the lower of which serves as obturator for the lower aperture of the cannula, while the other secures the upper end of that instrument. Intelligent use of the cannula presupposes, of course, an approximate knowledge of the location and length of the stricture. Any food remnants or mucus that may be in the esophagus must be removed by irrigation before the sound is inserted. The use of this cannula is perhaps most suitable in cases of carcinomatous stricture which do not ulcerate and are not yet too far advanced. Extreme care is imperative, because a certain amount of force has to be employed. It goes without saying that lesions must be rigidly avoided. As soon as the cannula lies firmly in the stricture, the silk threads are detached from the clamp and the sound loosened and withdrawn by rotary and lateral movements. The free ends of the threads are conducted out at the angles of the mouth and securely fastened behind the ears.

Surgical Treatment.—Aside from the treatment by dilatation, in cases of carcinoma of the esophagus surgical intervention must be considered. Czerny was the first to carry out resection in the cervical section of the esophagus, in 1877, with favorable results. Since then the operation has been repeated about twenty-five times, unfortunately with but a small measure of success. The immediate operative mortality is at least 36 per cent. The final results are likewise bad, because in most cases recrudescence occurs sooner or later. Operative cases must be very critically selected. All the neighboring organs which are involved must be removed, as far as possible, and the esophageal operation must be preceded by gastrostomy. Carcinomata in the thoracic section of the esophagus may be attacked with the aid of intracheal insufflation of ether (Meltzer and Auer), but the results so far have been bad. Carcinoma of the cardia, however, where the lowest part of the esophagus can be connected with the stomach after resection of the carcinoma, offers a more favorable proposition.

Alimentation.—In all cases alimentation by mouth should be maintained as long as possible. The diet should be soft, liquid or pulpy, according to the narrowness of the stricture. Since the stomach is usually healthy, it does not matter so much what kind of food is administered, but rather that the food be given in a

form which facilitates deglutition. As the stricture increases, the problem of nutrition becomes more difficult. It is, of course, absolutely necessary that the food be not excessively spiced or acid, in order to prevent irritation of the carcinomatous tissue. Swallowing a tablespoonful of olive oil or sweet almond oil, or the yolk of an egg, previous to the ingestion of food, for its lubricating effect upon the stricture, is to be recommended. When it becomes necessary to abandon mouth feeding entirely, the patient's spirits and to some extent his strength may be sustained by nutritive enemata (see page 243).

Should food remnants, mucus, blood and pus accumulate above the stricture, the esophagus should be frequently irrigated by means of a soft-rubber stomach tube or hollow sound, using either warm water, salicylic acid (1:1000), silver nitrate (1:1000) or thymol (1:2000) solution. Even when there is no accumulation of food remnants, it is well to let the patient drink small quantities of alkaline mineral water both morning and evening.

Aside from these measures, the treatment of carcinoma of the esophagus can only be ameliorative, in the endeavor to relieve the symptoms as far as possible. For this purpose morphin is administered subcutaneously and internally, eucain or cocain solution injected with the esophageal syringe (Fig. 68), or tablets of cocain 0.02 Gm. ($\frac{1}{2}$ grain), anesthesin or orthoform swallowed. The following prescription is often useful:

	Gm. or Cc.	
R—Morphine sulphatis,		
Cocaine hydrochloridi	ss	0.0025 gr. $\frac{1}{2}$
Antipyrini		0.01 gr. $\frac{1}{4}$
Saccharini	3	0.3 gr. v
Misce et ft. tab. no. i.		

Sig.—One tablet several times daily

In the presence of pains and spasms, atropin may be administered to advantage, both subcutaneously and by mouth. In order to restrict ulceration, excoriation, and secretion of mucus, the mineral waters may be prescribed, or solutions of bicarbonate of sodium, hydrogen peroxid (1 to 2 per cent., one teaspoonful every hour), silver nitrate (1 per cent.), or epinephrin (5 to 10 drops of the 1:1000 solution in a teaspoonful of water).

Cauterization of very prominent parts of the tumor is permissible under certain circumstances, but of course is not attended with permanent results. Radium and mesothorium emanations, and the Roentgen ray, may be tried (page 550), although permanent results from this treatment have not so far been reported.

As to the treatment of cicatricial stricture, it may again be repeated that the sound can and should be used much more energetically and persistently than in the carcinomatous form, very good results having been thus obtained. Carefully selected, non-irritating, concentrated diet is an essential item. Dilatations

above a cicatricial stenosis should be irrigated when they contain stagnant food remnants. Manifestations of irritation in the stricture should be prevented by diet, mineral waters, and liberal doses of oil. When the stricture is very narrow, gastrostomy can be performed earlier than in carcinoma, owing to the possibility of continuing the sound treatment and dilating a deep-seated stricture from the stomach.

DIVERTICULA.

Traction Diverticula.—Traction diverticula of the esophagus are funnel-shaped or sac-like eversions resulting from traction from without. Inflammatory processes in the neighborhood of the esophagus, and especially caseous bronchial and tracheal glands which are adherent to it and undergo cicatricial shrinking, pull upon the esophageal wall, everting it to a certain extent. Fibrosis of the lymph nodes situated opposite the level of the bifurcation of the trachea exerts traction upon the wall of the esophagus, forming minute pouches. These diverticula may be 1 to 1.5 centimeters deep, and 7 to 8 millimeters wide at the entrance; the inner wall is smooth, and the mucous membrane usually normal, though in the deep parts it is sometimes cicatricially contracted, blackish or ulcerated. Traction diverticula, as a rule, run a course without symptoms, but when inflammatory processes occur in the neighborhood they may perforate and cause grave or even fatal complications by infection of the mediastinum. Should dysphagia occur, the diagnosis can be made with the esophagoscope and the Roentgen ray (see Chapter V).

Treatment.—Should there be no symptoms, treatment of traction diverticula is usually unnecessary; all that is required is care in diet, the patient being provided with well minced food of a bland nature. Sounds should not be used.

Pulsion Diverticula.—Pulsion diverticula may be caused by pressure from within the lumen of the esophagus. Among etiologic factors are: impaction and pressure of foreign bodies and solid morsels in the larynx, hasty swallowing of food, and ingestion of very hot food. It may be assumed, however, that these factors alone are not sufficient to cause an eversion of the esophageal wall in the absence of any congenital or perhaps even continuous trauma. In this connection fetal development is of importance, as many observations point to the possibility of these diverticula having developed from remnants of the internal fetal sulcus. They are situated exclusively at a point in the esophagus beginning behind the pharynx, opposite the cricoid cartilage, at the posterior wall, more often on the right than on the left side. This is the point where the external longitudinal muscular layer of the esophagus is least developed. The affection develops very slowly; probably it is often many years after initiation of the process before there are any symptoms.

The size of the diverticula varies; some are no larger than a hazelnut; others measure 13 centimeters in length by 5 centimeters in transverse diameter. These large diverticula hang between the esophagus and the vertebral column, with more or less extensive inosculation. The majority of the patients are between fifty and sixty years of age.

Symptoms.—The symptoms develop as slowly as the affection itself, commencing with slight difficulty in swallowing and a slight pressure sensation in eating. Frequently there is also eructation after meals and vomiting of small quantities of the ingested food. As the diverticula increase in size, the symptoms become more pronounced. The food glides into the diverticulum, which, according to its fulness, presses against the lumen of the esophagus, interfering with the passage of other food to the stomach. The diverticulum is very rapidly filled, causing the food to accumulate in the pharynx, whence it is brought up and vomited in a short time. Some patients are able to press the food down by inclining the head or applying digital pressure to the side of the neck where the diverticulum is situated. These manipulations, however, cease to avail with the increasing compression of the esophagus, so that finally no food will reach the stomach at all. Now appear the sequelae of chronic starvation: pronounced emaciation, hunger, thirst, and inanition. When the food stagnates in the diverticulum for a considerable time, it decomposes, ferments and putrefies.

It is a matter of diagnostic importance that these food remnants contain no hydrochloric acid, but often lactic acid and frequently large quantities of long microorganisms, as revealed by the microscope. There is often a strong, disagreeable *fetor ex ore*.

Large diverticula often occasion swellings of the neck which can be reduced by pressure.

Pressure of the diverticula upon neighboring organs, vessels, nerves (recurrent laryngeal), and trachea, is responsible for grave cardiac manifestations and dyspnea.

Diagnosis.—The esophageal sound will always glide into the diverticulum when the latter is comparatively well filled; when empty, the sound may glide into the esophagus. This is of diagnostic importance in cases where the findings of the sound vary. The diagnosis is aided by the diverticular sounds of Leube and of Starck. The esophagoscope is likewise of importance for purposes of diagnosis, as it enables the physician to observe the opening of the diverticulum. The Roentgen ray will definitely locate the diverticulum (Plate XI, Figs. 3 and 4).

Prognosis.—The usual course of the affection is for a time favorable, inasmuch as it develops slowly and often extends over decades, even in advanced stages, provided the patient conducts himself correctly. It is, however, always a grave condition, from which many patients die of inanition or lead an agonizing existence.

Treatment.—Fortunately, modern surgery has materially improved the formerly cheerless chances of these sufferers. The best method for permanently curing the affection is extirpation of the diverticulum. This operation has lost much of its danger, and a large number of operated cases have been reported as having taken a favorable course.

Should operation, for whatever reason, not be resorted to, the sound treatment is to be instituted. In some cases it may be possible to press back the protruding entrance of the divertic-

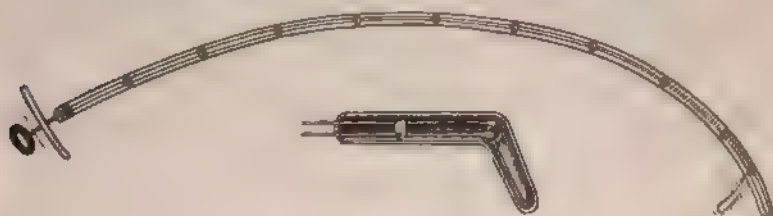


FIG. 76.—Diverticular sound. (Leube.)

ulum, causing it to contract. By this means the food will be aided in reaching the esophagus. As it is difficult in these cases to enter the esophagus below the diverticulum with a straight sound, a way out of the difficulty would be to deflect the lower end of a soft-rubber stomach sound and to manipulate the deflected end. This may sometimes result in reaching the eccentrically situated esophageal entrance.

The so-called diverticular sounds are constructed with this object in view. The one devised by Leube (Fig. 76) is an ordi-



FIG. 77.—Diverticular sound. (Starek.)

nary stomach tube, with a peculiar end-piece of German silver wire, flexible and hinged. This end-piece is inserted into the soft-rubber stomach tube. By means of a ring which protrudes from the upper end of the tube it is possible to make traction upon the movable point of the nozzle, deflecting it anteriorly together with the lower end of the tube. The entire instrument is inserted beyond the cricoid cartilage, the point is deflected anteriorly, and the sound is pushed down along the anterior wall. In this way it is often possible to enter the esophagus below the diverticulum.

Starck's sound (Fig. 77) is a powerful steel spiral with a metal core. At its lower end there is a thread upon which a straight or bent metal continuation of any size may be screwed. The deflected end is intended to facilitate penetration into an eccentrically situated esophageal entrance. Starck has also devised a hollow sound upon which the various metal continuations may be screwed, so that in case the attempt at penetration succeeds the sound may at once be used for artificial feeding.

The esophagoscope should be used as often as possible for the purpose of finding the esophageal entrance, sounding, and subsequent artificial feeding. The sound-treatment will, of course, be attended with the greatest success when the technic can be so arranged that the patient learns to insert a hollow sound himself and use it for alimentation.

FOREIGN BODIES IN THE ESOPHAGUS.

Obstructions due to foreign bodies in the esophagus are of frequent occurrence. Thus, a mere morsel of solid food may be arrested in a stricture, the existence of which has possibly never been suspected. But all kinds of objects may occasionally find their way into the esophagus, such as artificial teeth, coins, fragments of bone, children's toys, etc. When such an accident happens, if the object swallowed (or half-swallowed) was round and smooth, morphin is to be administered, and a thick sound inserted if possible. The purpose is (1) to determine the site of the foreign body, which may have passed into the stomach, though the patient still has the sensation of its sticking in the throat; (2) to carefully and gently push the foreign body into the stomach, so that it may be evacuated in the natural way, since smooth objects do not cause any injury to the mucous membrane. It should also be remembered that small objects may sometimes be recovered by means of an energetic emetic. If a pointed, sharp or cutting object has lodged in the esophagus, morphin should be administered, but the only admissible use of the sound under such circumstances is to establish by very careful manipulation the point of impaction. Sounding is forbidden when artificial teeth have been swallowed; these can in most cases be easily located by the Roentgen ray (see Chapter V).

If the foreign body is of such form or substance, even though it be round and smooth, that it cannot be evacuated by way of the intestine, it must be extracted. With the aid of the esophagoscope an effort may be made to loosen the object and to catch it with suitable instruments, forceps, coin-catchers, etc. Should this method fail, esophagotomy must be done. If the foreign body were allowed to remain in the esophagus it would cause decubital ulcers, hemorrhages, and perforations. Pointed or hooked objects may even cause lesions of the mucosa when extraction is performed

under the guidance of the eye. Should lesions occur during extraction, they can be easily healed by touching them with a 10- to 15-per-cent. solution of silver nitrate in the esophagoscope, and giving morphin if necessary (Fig. 64).

Foreign bodies which are arrested at the esophageal entrance are comparatively easy to extract. Occasionally, at least in children, they can be caught with a deeply inserted finger or with bent pharyngeal forceps.

OTHER CAUSES OF ESOPHAGEAL STRICTURE.

Thrush.—It has already been mentioned that thrush is capable of completely occluding the esophagus. Should this cause considerable difficulty in swallowing, an attempt may be made to render the esophagus permeable with a sound. As a local antiseptic, borax (in a 3-per-cent. solution, one tablespoonful every two hours) seems to have a favorable effect.

Compression of the Esophagus from without can take place, aside from diverticula, by aneurysms, tumors of the mediastinum, lungs or vertebral column, swelling of the mediastinal and bronchial lymph glands in tuberculosis or leukemia, and peri-esophageal abscesses. These may frequently be made out by means of the Roentgen ray (see Chapter V).

Spasm of the Esophagus. Spasms of the esophagus and cardia, caused by spastic stenosis, are a very frequent and practically important affection (see Cardiospasm, Chapter XVII). Any such spastic contraction renders the esophagus impermeable at some point. The spasm is of varying intensity and duration, and may be accompanied by more or less pain. The attacks recur at intervals, perhaps for years, or they may disappear as suddenly as they appeared. They occur in hysterical and neurasthenic individuals, being induced by emotions of all kinds. The esophageal spasm may be merely the expression of an isolated hyperesthesia of the esophageal mucosa, or it may be a symptom of disturbance of the vegetative nervous system (see page 387). There are also reflex spasms, caused by other affections and particularly by inflammations of the esophagus; gout and arteriosclerosis seem to favor their occurrence. Esophageal spasm occurs oftener in the female than in the male, especially at middle age (see page 137).

Symptoms.—Esophageal spasm manifests itself principally in disturbed deglutition. A swallowed morsel is arrested in the esophagus, and the patient experiences a sensation of contraction and pressure in the chest, which is often intensely painful. The paroxysms do not recur often at first, but the cyclic form tends to increase in frequency, always, however, observing certain intervals. This form often passes into a chronic stage in which there is dysphagia whenever food is taken. After having persisted for years,

the spasms not infrequently disappear without any assignable cause or therapeutic endeavor. The pathologic picture may therefore be one of considerable variation—which is diagnostically important. Oftentimes patients have less difficulty in swallowing solid food than liquid. In other cases the spasm occurs only with the first bite, after which the ingestion of food proceeds without difficulty. Concomitant manifestations, such as pains, palpitation, fear, dyspnea, are often very considerable. The esophageal sound meets with an obstacle at some point in the esophagus, which may disappear after a little while and reappear at a lower point. The spastically closed entrance of the stricture is distinctly visible through the esophagoscope. The Roentgen fluoroscope characteristically illustrates the arrest of the opaque bismuth meal and its lightning-like disappearance.

Treatment.—Provided the diagnosis of spastic stenosis is established, the first question, of course, is the etiologic treatment—treatment of the underlying affection (neurasthenia, hysteria). In neurotic patients the psychic treatment is always of great importance. If treatment of the underlying cause should fail, sounds will have to be used, preferably those of the largest caliber. One single application of the thickest sound, coupled with dilatation, often has a curative effect. In any case, regular application of the larger sounds should remove spasm in time. Very good results have often been achieved by direct dilatation of the spastic cardia or of the spastic part of the esophagus by means of the balloon sounds devised by Sippy and that of Myer (see page 369). The sound is introduced into the stomach or below the esophageal stricture, inflated, and withdrawn through the cardiac or esophageal stricture, or it is allowed to remain for some time inflated in the stricture. Entrance of the thick sound into the stricture is often facilitated by having the patient swallow. There is no need of force, as the mere presence of the sound above the stricture is often sufficient to relieve the spasm. When in grave cases the question of nutrition is involved, the use of the hollow sound is advisable in order to combine artificial feeding with the procedure. The application of the galvanic current has oftentimes a very favorable effect. For this purpose the galvanic gastric sound is inserted into the stricture, with the anode in the esophagus and the cathode at the nape of the neck, the current being allowed to pass for a few minutes. The combined stomach tube and electrode of Stockton (Fig. 30) has given good results. Galvanization from the outside may also be beneficial; the cathode is placed at the side of the larynx and the anode against the nape of the neck. Closure of the cathode will cause irritation of the vagus, under the influence of which regular deglutition will follow. In the presence of very pronounced hyperesthesia it is advisable to employ anesthetics in the form of cocain, eucain, anesthesin, or orthoform (see page 270).

Benzyl benzoate has recently been used with benefit in cases of spasm of the esophagus (see page 276).

Dilatation of the Esophagus.—Pathologic dilatations of the esophagus are either diffuse (uniform) or partial. The entire length of the esophagus is rarely involved. The favorite seat is above an organic stenosis, but this does not signify that every stricture is followed by dilatation, for the musculature of the esophagus is strong enough to rapidly throw out food, mucus and sputum which collect above even pronounced strictures. It is only when the musculature weakens that a part of the esophageal wall is dilated, less often the entire section above the stricture. The mucous membrane in the dilated area is inflamed, and the stagnant food remnants exert pressure upon it; frequent vomiting, eructation, and offensive *fetor ex ore* are among the manifestations.

A dilated condition of the esophagus without any anatomic obstacles is clinically and therapeutically more important. These cases are now well known clinically. There is usually a uniform fusiform dilatation which extends upward from the diaphragm. The esophagus is cystically distended, the greatest diameter of the sac being immediately above the diaphragm. In some cases the distended part resembles an elongated ellipsoid. The musculature in the dilated area, notably the layer of circular fibers, is usually hypertrophic. The mucous membrane may be normal, but usually it is hypertrophic and sometimes inflamed, covered with papillary proliferations, or eroded.

Etiology.—Etiologically, spasms of the cardia or of the lower section of the esophagus are chiefly to be considered. Further etiologic factors are atony of the esophagus, with subsequent distention and secondary cardiospasm. Trauma has also been held responsible, and it may well be imagined that a trauma may lead to a spasm, or atony or paralysis of the esophagus. Organic affections of the central nervous system or of the vagi may likewise cause disturbances in the innervation of the cardia and esophagus. The affection occurs principally in neurotic patients, and is about equally distributed between the two sexes. It occurs between the ages of fifteen and forty. Experience confirms the necessity for examining for gastroptosis in every case of dilatation of the esophagus, as the latter may be due to kinking from sagging of the stomach.

Symptoms.—The symptoms, especially in the initial stage, resemble those of spastic stenosis described above. In the pronounced pathologic picture there is this prominent fact to be considered, that the food can be swallowed but not conveyed to the stomach; the sensation is as if the food were arrested immediately before reaching the stomach. For this reason the manifestations are often referred to the stomach itself, the associated symptoms, such as pressure and pain, being localized

in the region of the epigastric angle. According to the development of the sac, the symptoms vary. It is easy to understand that a large, well-filled sac exerts great pressure upon the neighboring organs, the heart, diaphragm and lungs, causing corresponding disturbances. Oftentimes patients are awakened from sleep by violent paroxysms of coughing and suffocation, especially when in the horizontal decubitus the sac is evacuated upward and part of the evacuated mass invades the larynx.

Diagnosis.—All these manifestations, considered in conjunction with those of cardiospasm and esophageal spasm, point to the correct diagnosis. The latter is assured by examination with the sound, which can either not pass into the stomach at all, or only with difficulty. In this connection, the changing chemism of the material thus obtained is important, particularly when the sound once in a while happens to enter the stomach and gastric contents can be withdrawn. Aside from the sound, the esophagoscope is of diagnostic service, as the dilated esophageal wall presents numerous folds and ridges of mucous membrane. Furthermore, Roentgen-ray examination (see Chapter V) will serve to establish the exact diagnosis by showing with the aid of bismuth the dilated area. The condition of the mucous membrane is established by the esophagoscope.

Prognosis.—The prognosis is not so unfavorable as in diverticula. Cases which are not too pronounced or too old can unquestionably be cured or improved. Even in considerable dilatations of the esophagus it is possible in most cases to maintain the state of nutrition sufficiently to prevent a fatal termination.

Treatment.—The treatment depends upon whether spasm, atony or other organic change (esophagitis) has given rise to the dilatation. When there is spasm of the cardia, it is clear that the spasm has to be treated as such, in the manner described under that head. It should be added, however, that those conditions of cardiospasm which occur as sequelæ to chronic inflammatory processes of the mucous membrane are not suitable for sound treatment. Such organic changes of the mucosa might be exacerbated by sounding, thereby increasing the inclination to spasm. In this secondary spasm, therapeutic measures are often very successful. The patient is to have physical and mental rest, and if necessary be enjoined to keep long hours in bed. All kinds of stimulants (alcohol, coffee) must be strictly avoided. The diet should be absolutely non-irritating. Moist packing of the chest, packing of the body, warm baths and pine needle baths are often beneficial. In the presence of highly irritating conditions and considerable anxiety, regular small doses of opium and bromid (tincture of opium 0.3 Cc., potassium bromid 0.5 to 1 gram) or morphin should be prescribed. Papaverin and benzyl benzoate are both valuable (see page 276). Daily irrigation of the dilated

esophagus will no doubt improve the condition. Should the spasm nevertheless persist, local treatment of the cardia should be instituted. After irrigation of the esophagus, the application of a 3- to 4-per-cent. eucain solution to the cardiac region will serve to reduce the irritability of the cardia. Galvanization of the cardia also has a soothing effect. This can be carried out with the ordinary stomach electrode or a tube electrode devised by Stockton (Fig. 38). A very weak galvanic current, 0.5 to 1 or 2 milliamperes, is applied, according to the sensitiveness of the patient.

When there is a purely nervous spasm of the esophagus or cardia, the treatment described under *Cardiospasm* is to be applied to relieve the stricture. It may be again pointed out that dilatation of spastic stenosis with the balloon sound, as described on page 497, has often been attended with brilliant results, provided, of course, the cardia was at all permeable.

Among the general measures which hold good for all kinds of fusiform dilatations of the esophagus, irrigation of the dilated canal ranks first. The sac is irrigated daily after the manner of gastric lavage, preferably in the evening before going to bed. In order to prevent irritation of the stomach and intestine, it is important to see that as little as possible of the decomposed mass enters the gastro-intestinal canal. The best plan is for the patient to acquire the technic of the irrigation himself, which is not a rule a very difficult matter. Good olive oil and sweet almond oil, flowing into the esophagus after irrigation, will reduce irritability. Another important point is physical and mental rest. Patients whose condition is run down from lack of nourishment urgently require prolonged rest in bed. The ingestion of plenty of good food is of great importance. During the first two or three weeks nourishment is best administered by the sound in order to eliminate deglutition, but for reasons above cited this is impossible in the presence of inflammatory changes in the esophagus. In these cases rectal alimentation in the beginning of the treatment, continued for several days, would be indicated (see page 24). The food taken by mouth should be absolutely non-irritating, high in calories as possible, and not too abundant. General speaking, soft and pappy foods should be given, above all milk, fat, cream, oil and butter. Meat and vegetables, prepared in purée form, are of course permitted.

The atonic form of dilatation is often favorably influenced by mild gymnastics, hydrotherapy, and breathing exercises, owing to their effect upon the peristalsis of the esophagus.

Congenital Dilatation.—Congenital dilatation is a partial dilatation of the esophagus which occurs immediately above the point where the esophagus passes through the diaphragm, involving but a small section of it. This is the part which Luschka has termed the *manyplic* ("Vormagen").

Anal Stricture. Congenital stricture may assume any of the following forms:

1. Simple blind termination of the esophagus, which is a very rare occurrence. The existing part of the organ is a sac, tapering at the lower end and resembling a diverticulum. This cul-de-sac communicates with the cardia by a thin cord.

2. Blind termination of the upper end of the esophagus. The lower two-thirds of the developed esophagus communicates with the trachea, permitting the trachea to be reached by the finger. The two parts of the esophagus may communicate by a thin coat, or this connection may be absent.

3. The rare occurrence of complete obliteration of the normally developed esophagus at or below the level of the bifurcation of the trachea.

Infants with this form of congenital stricture are often afflicted with other malformations and nearly always asthenic. They are unable to swallow at all, or the milk returns through the nose. The occlusion of the upper section of the esophagus can be verified by the introduction of a sound. Usually these infants die soon. Operative intervention, which has been attempted, has proved futile. It might be possible to invigorate these patients first by the aid of gastrostomy and resort to operation later.

4. Membranous occlusion of the esophagus. This consists of a thin, transparent membranous or annular fold; very few locations on the esophageal mucosa are subject to this deformity, which, when it occurs, jeopardizes the permeability of the esophagus. When the prognosis is favorable, operative intervention may be indicated.

5. Simple strictures which, according to the anamnesis, have developed from infancy and anatomically exhibit a perfectly normal condition of the esophageal mucosa and musculature, without any cicatrization. These strictures often do not cause any trouble until the time comes when coarser food is taken. By judicious dieting it is possible to preserve life for years, and in these cases treatment is distinctly hopeful.

Rupture, Malacia, Perforation, Hemorrhage.—Rupture of a healthy esophagus may occur from grave thoracic trauma. Spontaneous rupture is exceedingly rare, but may occur in a perfectly healthy esophagus from sudden extreme exaggeration of the intra-abdominal pressure in violent vomiting or forcible contraction of the pharyngeal musculature caused by a large, hard morsel of food. Esophagomalacia also undoubtedly plays a rôle in spontaneous rupture. Esophagomalacia, like gastromalacia, often occurs after death as a result of the action of the acid gastric juice upon the esophageal membrane; or it may occur shortly before death, when, in the presence of greatest debility or unconsciousness (meningitis), the patient is unable to prevent regurgitation of gastric contents into the esophagus. In either case the esophageal mucosa, not being properly supplied with sufficient blood, can become softened and

digested. Evidently, however, esophagomalacia may in exceedingly rare cases occur in otherwise healthy individuals, when in some way or other a possibility has been created for the entrance of acid gastric contents into the esophagus. Predisposing factors are atony of the cardia and esophagus, habitual vomiting, hyperacidity, tendency to eructation and vomiting after an abundant meal at the height of digestion, prolonged shaking of the body (as in horse-back or automobile riding after meals). Esophagomalacia nearly always selects confirmed smokers and drinkers. As a rule the rupture occurs immediately above the cardia in the shape of a longitudinal tear; a circular tear has been observed only once.

Rupture is, of course, always followed by the gravest consequences. According to the reported cases which have been verified at autopsy, violent pain is experienced at the place of rupture at the moment it takes place, rapidly followed by the gravest manifestations—terrifying fear, dyspnea, collapse, pain, hematemesis. Deglutition is usually not interfered with. Rapid development of cutaneous emphysema, spreading from the face and neck over the entire body, is of diagnostic importance. The next manifestations are pneumothorax, pyothorax, and all the grave symptoms of an infected mediastinum. Death usually occurs within twenty-four hours.

Hemorrhage in the esophagus may occur in the course of ulcerous processes, carcinoma, corrosions, and in ruptures. The gravest and most important esophageal hemorrhages are those from the varices, which are found mostly in the lower third of the esophagus, above the diaphragm. The veins of the esophagus communicate with the portal vein, and this explains that stasis in the portal circulation leads to formation of varices. These varices are especially found in cases of hepatic cirrhosis, syphilitic affections of the liver, thrombosis of the portal vein and the mesenteric veins. But they are not always dependent upon portal stasis. These esophageal varices may attain to the diameter of a lead-pencil. A diagnosis of varices can hardly be made, because the blood vomited from the esophagus may just as well come from the stomach. The hemorrhage is usually very profuse. (See page 592.)

Esophageal hemorrhage is treated by means of the ordinary styptics (ice, injections of gelatin or ergotin); absolute rest; saline hypodermoclysis; 10-per-cent. sodium chlorid solution intravenously. The administration of hemostatic serum is beneficial if the coagulating power of the blood is below normal (see Chapter XXVI).

NEUROSES OF THE ESOPHAGUS.

Hyperesthesia. Hyperesthesia of the esophageal mucosa occurs in hysteria and neurasthenia, in conjunction with spasm of the esophagus and cardia. It is also a concomitant manifestation of

various affections of the esophagus (inflammations, ulcers, carcinoma) and is very frequently found in gastric affections (hyperacidity, hypersecretion). The symptoms of hyperesthesia vary considerably. There are often pains in swallowing, together with spasms. Sometimes there are unpleasant sensations in the esophagus when no food has been ingested—such as burning, pressure, and spasm, which may disappear during a meal. The seat of these symptoms may be the entire esophagus or a few sections of it. A similar disturbance is the so-called *globus hystericus*, but according to present views this sensation may also be present without hysteria. Pyrosis also involves hyperesthesia of the esophagus, as a consequence of the gastric contents moistening the esophageal mucosa; or the hyperesthesia may be purely neurotic without any regurgitation of gastric contents. In order to make a diagnosis it must be established by the sound whether there is a free passage, and this procedure may sometimes cause pain. The esophagoscope will reveal any anatomic changes. As a rule the affection is very obstinate.

Treatment.—The treatment is principally dependent upon the etiology. As to local treatment, a silver nitrate solution (0.2 to 0.3 per cent.), half a table-spoonful in a wineglass of distilled water, to be slowly taken in sips three times daily upon an empty stomach, has been highly recommended. The application of sounds may be useful. Rosenheim recommends a soft stomach tube covered with molten cocain (0.4 : 10 cacao butter), to be inserted and allowed to remain for about ten minutes. Eucain injections (4 per cent.) are also recommended. Other drugs which are sometimes beneficial are bromids, morphin and atropin.

Anesthesia.—Anesthesia of the esophagus is possible, but nothing definite is known on this point.

Paralysis.—Paralysis of the esophagus occurs as a part manifestation of central paralysis (apoplexy), bulbar paralysis, or multiple sclerosis, when the nuclei or trunks of the vagi become affected. Affections of the vagus also occur in the presence of mediastinal tumors. A preceding diphtheria may cause paralysis of the pharynx and esophagus. Paralysis has also been observed after grave trauma, and it is just possible that hysteria likewise plays a rôle in this respect.

Symptoms.—Impeded deglutition is the principal symptom, the food being arrested at some point in the esophagus, whence it can only be forced into the stomach by repeated swallowing efforts or the swallowing of a liquid. The sound meets with no obstacle in entering the stomach, being freely movable laterally. Liquids usually pass through the esophagus more easily than solid food. The affection is often difficult to distinguish from dilatation of the esophagus, to which it may easily lead.

Treatment.—Attention to diet, which should chiefly be liquid. If necessary, feeding through hollow sounds. Application of electricity (faradization of the esophagus), or strychnin injection 0.001 Gm. ($\frac{1}{80}$ grain).

Atony.—Primary atony of the esophagus may develop from a neurotic condition. In making the diagnosis it should be observed that liquids always pass without hindrance, while solid substances do not. There is no difficulty in passing the sounds. Organic affections can be excluded from the findings obtained through the esophagoscope. The differential diagnosis, however, between dilatation and atony is not always an easy matter.

Treatment.—General strengthening measures; hydrotherapy; arsenic; iron; faradization of the esophagus; adequate nutrition.

CHAPTER XVII.

MOTOR NEUROSES.

VAGOTONIA; SYMPATHICOTONIA; HYPERMOTILITY; PERISTALTIC UNREST; CARDIOSPASM; PYLOROSPASM; ERUCTATIONS; PNEUMOTOSIS; VOMITING; RUMINATION; REGURGITATION; PYLORIC INSUFFICIENCY; SINGULTUS GASTRICUS.

It is often difficult to establish a diagnosis of a purely nervous or functional derangement of the stomach—that is, to be certain that no organic disease is present. It is also difficult to ascertain whether or not the fundamental neurasthenia lying at the bottom of every neurosis of the stomach is the primary cause.

Neuroses of the stomach are differentiated from organic conditions by the one predominant symptom, referable to the motor, secretory, or sensory functions. This symptom has been termed "nervous dyspepsia." Some writers endeavor to draw distinct lines of demarcation between motor and secretory and sensory neuroses, and speak of nervous dyspepsia as a disease in itself, in which there may be present combinations of motor, secretory, and sensory disturbances, giving rise to purely subjective symptoms. Gastric neuroses develop principally in individuals of a nervous temperament—that is, in neurasthenics, hypochondriacs, and hysterical persons.

THE VEGETATIVE NERVOUS SYSTEM.

Functional derangement of the gastro-intestinal tract can be best understood by a study of the visceral nervous system. The innervation is supplied from three sets of nerves: first, the tonic or motor impulses, transmitted through the vagus; second, the inhibitory impulses through the sympathetic; and third, independent impulses, through the ganglionic plexuses of Auerbach and Meissner. These nerves can be distinctly stimulated and distinctly inhibited by certain medicaments. The first two sets of nerves are functionally antagonistic, and in correlation have been called by Langley the "vegetative nervous system"—a system that is self-governing and entirely independent of the impulses which originate in the cells of the cerebral cortex. They supply the smooth muscle and all the secretory glands of the digestive organs. Disturbance in the equilibrium of these nerves induces the classical conditions of hypermotility and hypomotility, atony, hypersecretion and hyposecretion, with their accompanying symptoms.

VAGOTONIA AND SYMPATHICOTONIA.

In contrast with the central nervous system, which serves the senses and the muscles controlled by the will, stands the vegetative nervous system—under which term we include all nerve fibers which are connected with organs having smooth muscles, such as the esophagus, stomach, intestine, liver, gall bladder, pancreas, blood-vessels, gland ducts, and skin, as well as the nerve structures which determine the activity of the glands of secretion. Besides these, certain organs are included in the system which have cross-striated muscles, such as the heart, cardia, pylorus, anus, and the muscles of the genital apparatus. One marked distinction between the central and the vegetative nervous systems is that the latter has ganglionic cells interposed in the course of the nerve distribution. This characteristic makes it possible for the anatomist and the physiologist to definitely separate the two great nervous systems. When vegetative nerves are stimulated at their origin, definite manifestations occur, but if nicotin be painted upon a ganglion between the site of stimulation and the periphery these manifestations cease at once. The functional manifestations of the central nervous system are unaffected by nicotin.

The vegetative nervous system embraces the sympathetic and related ganglia supplying the organs of involuntary bodily function independent of the central nervous system, and in addition the cranial and sacral nerves. One part of the system is supplied by the thoracic cord and the upper area of the lumbar cord, by way of the sympathetic ganglia, while another part originates in the medulla and the sacral segment of the spinal cord; the latter is called the autonomic or extended vagus system.

It will thus be seen that the vegetative nervous system consists really of two great systems—the sympathetic and the autonomic. The viscera are innervated by both. Both are under the control of the internal secretions. Electrical investigations show that manifestations caused by stimulation of the fibers of the sympathetic may be counteracted by stimulation applied to fibers of the autonomic, and *vice versa*. The two systems seem to be functionally antagonistic to each other. This is proved pharmacologically. Epinephrin acts solely upon the sympathetic system; its action is equivalent to stimulation of the sympathetic fibers. The autonomic system can also be influenced by drugs. The most important are atropin, pilocarpin, physostigmin, and muscarin. It is known that epinephrin flows continuously from the adrenals and that this internal secretion exerts a continuous influence upon the sympathetic nervous system. Eppinger and Hess believe that there may be an internal secretion, denominated by them "autonomin," which continuously stimulates the autonomic nervous system.

The autonomic nervous system stimulates motility, secretion, and

sensation, while the sympathetic inhibits them. When both systems act harmoniously and are properly balanced, normal bodily function results. If for any reason this functional equilibrium is broken, there results a condition of variation in tonus, depending upon which system is overstimulated—that is, a condition of either vagotonia or sympathicotonia.

From overstimulation of the autonomic nervous system we have the condition known as vagotonia. When the sympathetic system is overstimulated, sympathicotonia results. Certain drugs induce antagonistic action by stimulating both systems. The apposition of the two systems prevents acute transition of the functions of the visceral organs from one extreme to the other. It is quite possible that there exists in the central nervous system a center which controls the antagonistic action of these two systems. It is clear that a disturbance in either system will cause an increased or decreased tonus in the other, which may become the basis for the development of a pathologic condition.

Sufficient evidence has been adduced to show that disturbance of the vegetative nervous system plays a most important rôle, not only in initiating local gastro-enteric functional defect, but also in controlling and shaping the course of such defect to the point of profound organic change.

Excessive stimulation of the autonomic nervous system causes spasm of the circular muscles of the alimentary canal. This overstimulation may induce esophagospasm, cardiospasm, gastrospasm, pylorospasm, enterospasm, or proctospasm. Indeed, an excessive tonus and a spastic condition of separate segments of the gut can result in chronic spastic constipation. The typical vagotonic never has a dry mouth. Attacks of sweating are associated with many other conditions of increased tonus in other parts innervated by the vagus, such as nausea, vomiting, asthma, angina pectoris, and gastric crises.

In vagotonia there is increased tone, peristalsis and secretion of the stomach. The cow-horn stomach, with muscular tone, belongs to the vagotonic individual. Hypersecretion and hyperacidity are traceable to stimulation of the autonomic nervous system. Pain is a common accompaniment of this condition. Stimulation of the circular pyloric muscles is the underlying cause of pylorospasm. Esophagospasm and cardiospasm are both typical of vagotonia. Muscular spasm of the stomach induces an ischemia of the gastric mucous membrane, with loss of the antiferment, allowing the excess of hydrochloric acid to attack the mucosa, when accidental infection may lead to the formation of gastric ulcer. The initial lesion of peptic ulcer may be due to a disturbance of the vegetative nervous system, and this may be brought about by a disturbed hormone balance between the thyroid and adrenals. G. A. Friedman has proved the truth of this statement by animal experimentation;

after adrenalectomy, parathyroidectomy, or repeated injections of thyroid, pilocarpin or epinephrin, lesions, erosions or acute ulcers developed in the stomach.

Both diarrhea and constipation result from vagotonia. When the longitudinal muscle fibers are involved, diarrhea occurs, and when the circular muscle fibers are affected the result is spastic constipation.

The tender, palpable colon is a prominent sign of vagotonia. In many cases the small masses of fecal matter are covered with mucus. This increased secretory activity of the colon induces a condition that approaches mucous colitis. We regard mucous colitis as a secretory neurosis of the intestine, because atropin has such a markedly beneficial action upon it. The cause of the disease does not lie in the intestinal mucosa, for, though we call the condition mucous colitis, there is no inflammation of the colon. The cause undoubtedly is, in many instances, some disturbance of the autonomic nervous system. Many patients suffering from mucous colitis give a history suggestive of prolonged intestinal spasm. Spastic constipation is often accompanied by spasm of the anal sphincter so severe as to prevent defecation that would otherwise occur. The patient fears the pain.

Epinephrin stimulates the sympathetic nervous system as pilocarpin stimulates the autonomic. Numerous investigations have proved that there exists a pharmacologic antagonism between epinephrin and pilocarpin.

Atropin paralyzes the peripheral ends of the autonomic nerves, quieting the spastic intestine. The action of the drug is best observed in cases of increased tonus or vagotonia which have led to increased intestinal peristalsis.

Diagnostic Phenomena in Disturbances of the Vegetative Nervous System.—A classification of certain groups of diseases showing a predominance of vagotonia or sympathicotonia signs is not very difficult. Many of these signs and symptoms can be produced by decreased functioning of one of the systems, with overfunctioning of the other. The following are a few of the many tests for the recognition of vagotonia and sympathicotonia. Aschner's phenomenon (oculocardiac reflex) is produced by continuous pressure with the fingers on the eyeballs for one-half to one minute. In the normal person retardation in the pulse-rate follows, but rarely to a greater degree than ten beats to the minute. In vagotonia there is a marked slowing of the pulse during this procedure, and at times even a bradycardia.

Hering's phenomenon is evoked by slow, deep respirations. Normally the rate and volume of the pulse continue practically constant during deep inspiration and expiration. In vagotonia, inspiration causes an increase in the rate of the pulse and a decrease in the volume, while in expiration the pulse becomes slower and

increases in volume. On forced inspiration the pupils are dilated, and on expiration contracted.

The pilocarpin test consists in administering 0.006 Gm. ($\frac{1}{10}$ grain) of pilocarpin hypodermically. In sympathicotonia no salivation follows. In vagotonia extreme salivation and bronchial secretion occur.

Other tests, such as fat tolerance, carbohydrate tolerance, epinephrin sensitiveness, and cutaneous sensitiveness, have been suggested. This whole subject is of such great importance in the diagnosis and treatment of neuroses of the gastro-intestinal tract that I have tabulated some of the important symptoms and signs suggestive of derangement of the vegetative nervous system:

VAGOTONIA.	SYMPATHICOTONIA.
Miosis.	Mydriasis.
Accommodation spasm.	Accommodation paralysis.
Frequent winking.	Infrequent winking.
Epiphora.	Dryness of eyeballs.
Enophthalmos.	Exophthalmos.
Excessive perspiration.	Dryness of skin.
Ptyalism.	Dryness of mouth.
High gastric acidity.	Low gastric acidity.
Achylia.	Gastrorrhea.
Gastro-intestinal hypermotility.	Gastro-intestinal hypomotility.
Spastic constipation.	Atonic constipation.
Mucous colic.	No mucous colic.
Diarrhea, occasionally.	Diarrhea, rarely.
Bradycardia.	Tachycardia.
Low blood-pressure.	High blood-pressure.
Asthma.	Urticaria.
Cardiospasm, pylorospasm.	Gastric atony.
Enterospasm, proctospasm.	Intestinal relaxation.
Status lymphaticus.	Tonsils atrophic.
Gag reflex absent.	Gag reflex marked.
Eosinophilia.	Eosinopenia.
Clammy hands and feet.	Dry hands and feet.
Dermographia.	No dermatographia.
Aschner's oculocardiac reflex positive.	Aschner's oculocardiac reflex negative.
Pilocarpin test positive.	Pilocarpin test negative.
Increased fat tolerance.	Steatorrhea.
Graefe's sign positive.	Möbius' sign positive.

We recognize that a hypertonic state of the musculature of the stomach and intestine is a constant result in vagotonia. There is an overcontraction of the intestinal canal, which is manifested in the condition known as spastic constipation. Upon palpation the sigmoid may be felt as a thick cord. The anus is tight and contracted. These patients often complain of abdominal pain soon after meals. Partaking of cold liquids may induce a sudden attack of diarrhea, with excretion of a large quantity of mucus. The colics of enteritis membranacea have in their etiology a disturbance of the vegetative nervous system. (See Chapter XXXVI.)

Treatment.—In the treatment of all the neuroses of the gastro-intestinal organs, disturbance of the visceral nerves deserves careful

consideration. The results from medical treatment have proved eminently satisfactory. In hyperacidity, hypermotility, cardio-spasm, pylorospasm, enterospasm, spastic constipation, and enteritis membranacea, atropin or belladonna is our sovereign remedy—extract of belladonna 0.008 Gm. ($\frac{1}{4}$ grain) three or more times daily until the physiologic action of the drug is secured, the medication to be continued until the throat becomes dry, when the dose should be gradually decreased. Benzyl benzoate often relieves the spasm (see page 276).

In the treatment of low gastric acidity, gastric and intestinal atony, constipation, and gastroenteroptosis, pilocarpin is the best remedy. It stimulates the branches of the vagus which supply the gastro-intestinal organs, and is consequently vagotonic. Pilocarpin should be given in doses of 0.0015 Gm. ($\frac{1}{40}$ grain) three or more times daily until the physiologic action of the drug is secured. The annoying itching sometimes present is rapidly relieved.

Regulation of diet, hyperalimentation, education of the patient to a rational mode of life, hydrotherapeutics, gymnastics, electricity, massage, are all of value. The physician may exercise a profound influence over the patient's mental condition when he is able to do so; the progress of the case toward recovery will be much more rapid than it would otherwise be. The prognosis will depend largely upon the duration of the treatment, which, in the majority of cases, must be protracted.

HYPERMOTILITY (HYPERKINESIS).

"Hypermotility" is a term which designates an abnormally increased movement in the evacuation of the stomach, so that the viscus is often found empty soon after the ingestion of food. Hypermotility may occur in cases of achylia gastrica, the closure of the pylorus being defective on account of the diminution or absence of hydrochloric acid secretion; or it may occur with any other variety of pyloric insufficiency. It is a frequent accompaniment of duodenal ulcer. Cases of purely neurogenous hypermotility are rare. The diagnosis is established by means of a test breakfast or the Roentgen ray. Hypermotility does not often give rise to distressing symptoms, and consequently does not require any particular treatment.

PERISTALTIC UNREST OF THE STOMACH.

The complex of symptoms first described by Kussmaul, and attributed by him to peristaltic unrest of the stomach, does not often occur as a pure neurosis. The patients experience sensations of constant "gripping and moving" in the stomach and abdomen. When the abdominal walls are thin and the stomach more or less

ptotic, it is possible for the examiner to discern the actual peristaltic movements of the stomach. These movements are invisible through the abdominal wall when the stomach is in its normal position. A more or less rapid peristalsis is occasionally accompanied by rolling sounds which can be heard at some little distance from the patient. This condition is often present in stenosis of the pylorus. In making the diagnosis, mechanical obstruction about the pylorus must be ruled out.

Treatment.—The treatment consists in combating the cause as well as the general nervous symptoms present. In stenosis of the pylorus a gastroenterostomy is indicated. Excessive exertion, either mental or physical, must be carefully avoided. Nutrition should be regulated in order to avoid the ingestion of anything that might irritate the stomach; the food should be of a bland, semiliquid nature, and too great a quantity should not be allowed at any one time, for fear of overdistending the stomach. The evening meal should be light. The milk cure, combined with rest in bed, is worthy of trial. The direct local treatment consists of either cold or warm applications over the stomach, with lavage in the presence of dilatation and pyloric stenosis. Electric treatment in the form of either the galvanic or faradic current may be employed (see page 215). Sometimes confining the patient to bed and resorting to rectal feeding gives good results, owing to the physiologic rest thus afforded the stomach. A two weeks' course of nutrient enemata (see page 243) often results in complete recovery. The drug indications include the use of the bromids. Strontium bromid, 1 Gm. (15 grains) four times a day in water, or colelin phosphate, 0.01 to 0.03 Gm. ($\frac{1}{8}$ to $\frac{1}{4}$ grain) every two or three hours, may be prescribed for the relief of pain. Extract of belladonna, 0.01 Gm. ($\frac{1}{8}$ grain), sometimes affords great relief. For rheumatic patients acetylsalicylic acid 1 Gm. (15 grains) and bismuth subnitrate 1 Gm. (15 grains), three times a day, has proved beneficial. Vibratory massage over the left side of the tenth, eleventh and twelfth dorsal vertebrae has also given relief in this condition; it should be performed daily, the treatment lasting five minutes.

CARDIOSPASM.

Cardiospasm is a condition in which a spastic contraction occurs at the cardiac orifice of the stomach. The esophagus becomes closed up at its junction with the stomach at the moment of swallowing, so that it is impossible for either solids or liquids to enter the gastric cavity. Under normal conditions the cardia is capable of contraction and relaxation. The contractile force is situated in the cardia itself through the vagus, while the power of relaxation is controlled from the thoracic ganglia, whence the inhibitory impulses proceed to the cardia. During each act of swallowing, inhibi-

tory impulses pass from the sympathetic to the cardia, causing the latter to open to receive the bolus of food. In cardiospasm this inhibitory control is apparently absent, so that the cardia remains in a state of continuous contraction. Cardiospasm is probably due to an affection of the vegetative nervous system.

The term "achalasia," meaning absence of relaxation, has been suggested by Hertz as descriptive of the condition commonly known as cardiospasm. In achalasia a rubber tube filled with mercury will drop through the cardia into the stomach without the slightest difficulty, thus differentiating this condition from obstruction caused by a growth.

Symptoms. Examination of the cardia in this condition has revealed hypertrophy of the muscles and slight atrophic changes in the pneumogastric nerve. Cardiospasm, as a rule, starts suddenly during eating, and may pass off rapidly (acute cardiospasm); or it may persist for a long time (chronic cardiospasm). When the condition becomes chronic, patients while eating experience a sensation of pressure in the chest, which at times assumes the character of spastic pains radiating toward the bowels. The morsel of food is felt sticking in the esophagus, only to pass, after a time, into the stomach; or retching may cause its regurgitation into the mouth. When cardiospasm of this character continues for any considerable length of time, a loss in weight results, due to undernutrition. Retained food causes irritation of the esophageal mucous membrane, and the esophagus in chronic cases becomes dilated above the cardia.

Diagnosis. The diagnosis of cardiospasm is made by close observation of both subjective and objective symptoms. The objective examination consists in the introduction of a soft stomach tube or esophageal bougie, which, in the presence of cardiospasm, is grasped by the cardia and retained by the spastic muscular contraction. The spasm relaxes only after a period of waiting. Under any other condition, except obstruction by benign or malignant growth, the cavity of the stomach may be easily reached. This phenomenon is characteristic of cardiospasm. Dilatation of the esophagus is ascertained by the presence of undigested food remnants. Another feature of diagnostic importance is the fact that the so-called *second sound* of deglutition appears late or is often absent in cardiospasm (see page 350). Meltzer refers to the diagnostic importance of the inability to vomit. The diagnosis may be further confirmed by esophagoscopy and Roentgen-ray examination (see page 137). Roentgenograms (Plate XI, Fig. 1) taken shortly after the ingestion of the bismuth meal show the constricted cardia, with dilatation of the esophagus.

Delineator String.—Einhorn and Scholtz have devised a delineator string that casts a definite shadow on the fluoroscopic screen or on the plate, which has proved to be of great value in the diagnosis of

cardiospasm. It is made of very fine copper wires, sixty in a single strand, which is covered with braided silk; a metal ball is attached to one end of the string. This strand of copper wires offers no resistance whatever to spastic movements of the esophagus, being as pliant as a piece of cotton string. It is introduced into the esophagus in the same manner as the stomach bucket (see page 72). Under the Roentgen ray the shadow of the string appears more or less tortuous, according to the degree of esophageal spasticity present. Alterations in the outline of the esophagus from time to time are easily recognized by the roentgenologist in cases of intermittent

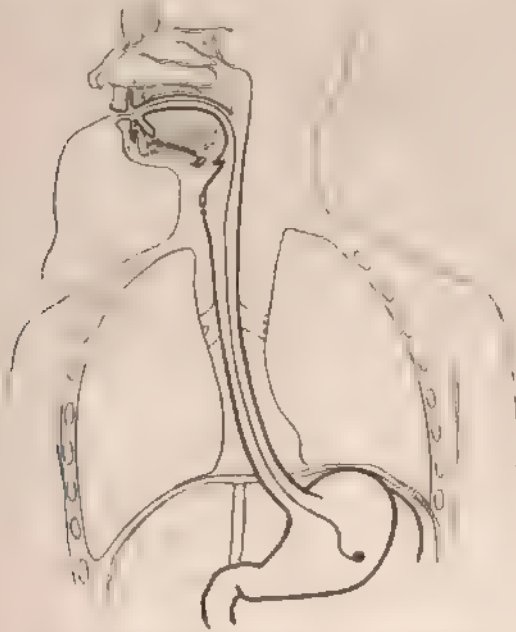


FIG. 78. Delineator string in normal individual.

cardiospasm. Once the delineator string has been introduced, it may be allowed to remain, so that the roentgenologist can make as many examinations as he pleases without subjecting the patient to the inconvenience of repeated ingestions of barium or bismuth. (Figs. 78 and 79.)

Prognosis.—The prognosis of cardiospasm is always uncertain. Acute cardiospasm occasionally disappears entirely, or reappears only at rare intervals. In chronic cardiospasm the prognosis is less favorable for complete cure, and the disease must always be regarded as somewhat serious because the sacculum above the constriction persists indefinitely.

Treatment.—The treatment of cardiospasm consists in the treatment of the neurotic conditions underlying it. The psychic factor of treatment is important, and patients must be reassured by the physician that the dread of swallowing which is always present may be dismissed. The patient should be prevailed upon to perform the act of deglutition several times without any food in the mouth before each meal, after which he should attempt to eat. His attention during meals should be diverted from his condition. Change of location, change in the usual habits of life, as well as a different arrangement of the meal hours, will sometimes be

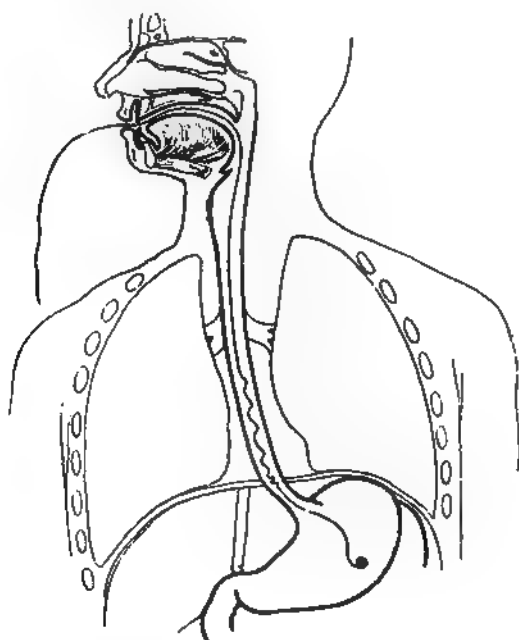


FIG. 79.—Delineator string in patient affected with cardiospasm.

accompanied by favorable results. All articles of diet apt to irritate the esophagus should be avoided. Patients must be instructed to eat slowly and to masticate their food thoroughly. Extremes of temperature in food and beverages should be avoided. Liquids containing much carbon dioxid, as well as acid or highly seasoned foods, are not well borne by patients in this condition. It is important, likewise, that the consistency of the food should be liquid or semisolid, though sometimes patients can swallow solid food to better advantage than liquid. A highly nutritious diet with as little bulk as possible should be the rule for these patients. Sometimes nutritive enemata are indicated, and may be given

for more or less prolonged periods of time, affording rest to the esophagus and cardia (see page 243).

The *oil cure* as recommended for carcinoma of the cardia (see page 552) should be employed in the treatment of cardiospasm. Mayonnaise and almond milk may be substituted for olive oil. Drug treatment, as a rule, has no direct effect upon the spasm. In selected cases, however, extract of belladonna has given fair results. Suppositories of atropin or eumydrin may be prescribed. Cases of mild cardiospasm quickly improve under the influence of a nerve sedative. Bromids in large doses are also indicated. In painful cases benefit has been derived from the administration of milk of almonds with the addition of anesthesin. Benzyl benzoate is very advantageous (see page 276).

Mechanical Treatment. Chronic cardiospasm should be treated along mechanical lines, and the cardia must be dilated by means of sounds.

Myer has devised a valuable dilator for the treatment of cardiospasm. The cardiac end of this instrument consists of, from within outward, first a rubber tube, one-fourth of an inch in diameter, closed at one end and at the other end continuous with the esophageal tube, next, extending for about six inches up and around this $\frac{1}{4}$ -inch tube, and made air-tight at each end, is a casing of thin rubber known as Penrose rubber tubing, which may be procured in three sizes—No. 1 and No. 2 for the large dilator and No. 2 and No. 3 for the small; and encasing the Penrose rubber tubing is a bag made of ordinary white silk with a diameter of about three centimeters (Fig. 80). The size to which the dilator can be distended depends upon the limitation offered by the silk bag. The outer covering of all is Penrose rubber tubing, securely fastened by means of silk at either end. A flexible mandrin consisting of a steel cable is used in introducing the dilator, and removed when the latter is in proper position. The dilating process is performed by means of a large glass or metal syringe such as is used in bladder irrigation. The syringe should be of at least 150 Cc. (3 v) capacity. Great pressure may be exerted, overstretching of the dilator being prevented by the silk bag or collar. When the sound has been introduced as far as the cardiac orifice, the rubber bulb is distended by water pressure from the syringe. The operation is to be repeated at weekly intervals.

Large esophageal sounds or bougies should be used, and left in position in contact with the stricture. It is better to begin with sounds of medium size and to increase the size as the stricture yields to the dilating process. The metal spiral sound of Crawcour (Fig. 71) has also been employed; owing to its pliability and weight, this sound very easily passes through strictures of almost any degree. The treatment by dilatation must be continued for a long period if satisfactory results are to be obtained. Esophageal lavage should

also be performed during the process of dilating the esophagus. Other methods of dilating the esophagus are fully described in Chapter XVI.

Electrotherapy. Internal galvanization of the stomach has been employed in a few cases of cardiospasm. Sometimes relief is only to be procured by chloroform narcosis.

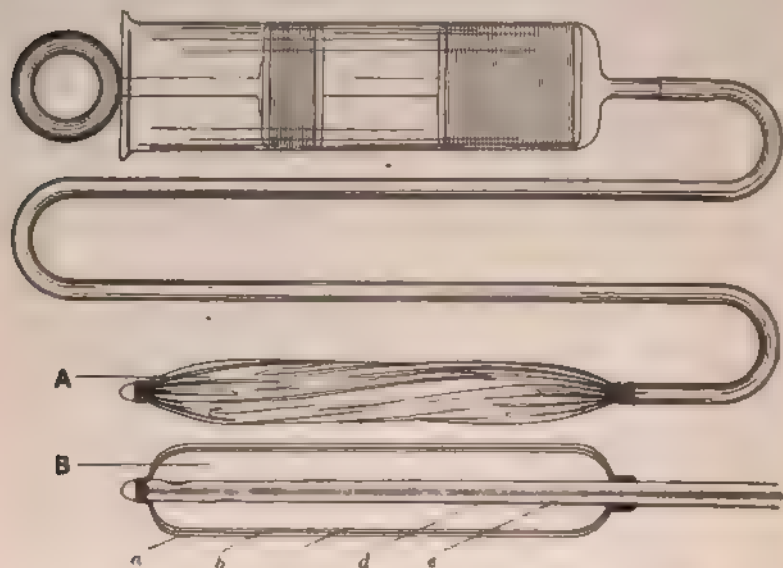


FIG. 80.—Mier's cardia dilator. A, deflated; B, inflated. a, outer rubber bag, b, silk bag, c, inner rubber bag, d, rubber tube, e, mandrin.

Surgical Treatment.—Surgery, too, has been employed. The cardia has been forcibly dilated directly from the opening made by gastrostomy. Divulsion of the sphincter of the cardiac orifice has been done. Willy Meyer reports successful treatment by thoracotomy and esophagoplication.

PYLOROSPASM.

Secondary pylorospasm is of comparatively frequent occurrence. Recent studies of the autonomic nervous system would seem to point to the probability of spasm of the pylorus being sometimes of purely nervous origin. The diagnosis may be established by exclusion of the usual causes of secondary spasm of the pylorus, such as carcinoma, gastric ulcer, duodenal ulcer, gall-bladder disease, appendicitis, and secretory disturbances. Pylorospasm from vagus overfunctioning may cause delay in emptying of the gastric contents, and this is usually associated with hypersecretion (see Chapter XXI). Pylorospasm occasions pain of greater or less

severity, together with increased gastric peristalsis and sometimes vomiting. Secondary pylorospasm may be caused by ulcer of the pylorus or duodenum, when the excessive acidity induces a spastic contraction—usually an hour or more after meals, at the time the acid enters the duodenum. This has been called by Moynihan "hunger pain" and by surgeons is regarded as diagnostic of duodenal ulcer. There are many other conditions, however, that will induce pylorospasm, and therefore "hunger pain" does not possess the certain diagnostic value which many would have us believe. We occasionally find pylorospasm in gastritis, duodenitis, cholecystitis, appendicitis, uterine disease, eye-strain, and anomalies of secretion. The Roentgen ray (see Chapter V) will often assist in the differentiation between primary and secondary pylorospasm. The symptoms of pylorospasm are found in cases of hyperchlorhydria (see Chapter XX). In a case of chronic appendicitis I was able to induce pylorospasm and epigastric pain by pressure over McBurney's point under the guidance of the fluoroscope (see Aaron's sign, page 773).

The differentiation between organic obstruction of the pylorus and pylorospasm is frequently quite difficult. One of the best methods of determining the patency of the pylorus is the use of the Einhorn duodenal bucket (Fig. 8). This is a small gold bucket, similar in shape to the stomach bucket, attached to a silk cord. The bucket is placed in a capsule and swallowed by the patient, and not withdrawn for several hours. On withdrawal, the contents of the bucket are examined for pancreatic ferments, and if these are found we are reasonably sure the bucket has been in the duodenum, thus proving that the pylorus is still patent. When the bucket has entered the duodenum the thread near it is golden yellow, due to the presence of bile. It is important that the stain on the thread extend only a short distance (10 to 15 centimeters). If one-third or more of the thread is bile-stained, this would indicate a regurgitation of bile into the stomach, and therefore forbid any conclusion regarding the passage of the bucket through the pylorus. The bucket will never reach the duodenum when there is an organic pyloric stenosis, while in pylorospasm it passes through (see page 479).

The diagnosis of pylorospasm¹ can be made with the delineator string (Figs. 76 and 77) just as the diagnosis of cardio-spasm is made (see page 394). It is better to introduce the delineator string in the evening and leave it *in situ* over night. The Roentgen-ray examination takes place the following morning. If the metallic ball is still in the stomach the obstruction is probably of an organic nature. Under normal conditions the metallic ball should be in the intestine and the course of the string presents a curved line accord-

¹ M. Einhorn and T. Scholz: Roentgen Ray Findings with the Delineator in Cases of Pylorospasm, Medical Record, November 27, 1920.

ing to the individual position of the organs. This line, though curved, is even, without any irregular zigzagging. If there is a spastic condition at the pylorus the Roentgen ray shows the delineator string irregular, corresponding to the degree of spasticity.

Treatment.—The treatment of pylorospasm consists in the removal of the underlying cause. This may require surgical intervention or, among other things, the application of heat, the administration of bromids and belladonna, codein, galvanization, and mild hydrotherapeutic measures. Stockton reports excellent results after the hypodermic injection of 1 Cc. (16 minims) of 1:1000 epinephrin solution. Papaverin hydrochlorid diminishes the tonus of the gastric musculature without exerting any influence on peristalsis. It is a valuable therapeutic agent in the dose of 0.03 to 0.06 Gm. ($\frac{1}{4}$ to 1 grain) three times daily. Benzyl benzoate in alcoholic solution is beneficial (see page 276). A bland, non-irritating diet should be prescribed, such as that recommended in the treatment of gastric ulcer, and only small quantities at a time allowed during the initial stages of treatment in order to avoid overdistention of the stomach.

The oil treatment, as outlined by Cohnheim, is valuable (see page 481). The oil should be taken on the fasting stomach. Hyperchlorhydria is frequently the cause of pylorospasm; the treatment of this condition is outlined in Chapter XX; atropin and eumydrin, together with the alkalis, are useful in pylorospasm due to hyperacidity. It has been maintained that the so-called congenital stenosis of the pylorus, or the pylorospasm of infants, is caused by marked hyperacidity or hypersecretion of gastric juice, secondary to congenital neurosis. An early diagnosis of the condition might possibly be obtained by examination of the gastric contents of the vomiting infant for hyperacidity, and many cases be thus saved from a fatal termination.

Einhorn's method of dilating the pylorus by means of a thin stomach tube and a small rubber bag should be employed if necessary. In the Einhorn pyloric dilator (Fig. 82, page 485) a small metal end-piece is attached to a thin rubber tube (8 millimeters in circumference and 1 meter long) bearing markings: I = 40 cm.; II = 56 cm.; III = 70 cm.; and 80 cm. Adjoining the metal end-piece and fastened to it and the tube is a tiny rubber balloon covered with silk gauze. The tube has a few perforations in the space covered by the balloon, and is connected at its upper end with a graduated glass syringe which serves the purpose of inflating the balloon with air.

Technic.—The pyloric dilator is introduced in the same manner as the duodenal tube (see page 501). After emptying the rubber balloon of its air contents (this is done by drawing the piston of the syringe outward), the cock is closed. The end-piece of the dilator is now dipped in lukewarm water and introduced into the pharynx of

the patient. The patient drinks some water, and the instrument moves into the stomach. It is now left in the digestive tract for several hours; or, better, it is swallowed before the patient retires, and left undisturbed overnight—for in pylorospasm it sometimes takes a long time for the apparatus to pass into the duodenum. In the morning the stretching is performed. Before doing this it is necessary to ascertain whether the dilator is in the duodenum. This is done by estimating the length of tubing within the digestive tract (it should be in as far as mark III, or 70 cm.); on drawing the tube slightly outward, it usually shows mark II within the mouth. The balloon is then inflated by means of the syringe. If the tube be now drawn forward there is a sensation as if the end of the instrument were held tight by something that drags along with it, not being able to escape it. It is not permissible to use much force. The balloon is then slightly deflated. This is repeatedly done until the end of the dilator by a slight pull passes through the pylorus. The syringe being graduated, one notes the number of cubic centimeters of air in the balloon during its passage through the pylorus. While the dilator is being drawn through the stomach no resistance is felt until the cardia is reached. Here the dilator should be entirely deflated and withdrawn—which is accomplished without trouble. Should, however, resistance be encountered at the introitus esophagi, the patient should swallow, and while his larynx moves upward the instrument is gently withdrawn without the application of any force.

NERVOUS ERUCTION (AEROPHAGY).

This condition is characterized by belching, which appears to be independent of the reception of food; it consists in eructations of air, accompanied by sounds which are audible at a considerable distance from the patient. The belching may persist for hours and in a few cases it has been reported to have kept up for days. The condition is one which affects chiefly neurotic individuals with or without gastro-intestinal disease. Such individuals are known to have a habit of eating or swallowing air (aerophagy). Neurotics suffering from digestive diseases sometimes experience trifling discomfort in the stomach which they attribute to an accumulation of gas, and in their efforts to obtain relief they expel whatever the stomach contains, whether "gas" or atmospheric air, and in the act swallow more air. When patients are suffering from some gastro-intestinal disturbance, the proteins or carbohydrates are apt to decompose, producing some little gas. The pressure induced by this gas is readily relieved by belching. The patient remembers the great relief thus obtained, and when he has a similar attack of gastric weight, pressure, distention or pain,

his first thought is to get relief by raising the gas from the stomach. An effort is required and in the effort the patient unconsciously swallows air, allowing it to come back quickly after each expulsive contraction of the stomach, and thus he becomes an aerophagic.

During an acute aerophagic attack, patients may suffer from dyspnea, tachycardia, and cyanosis. These are instantly relieved by introducing the stomach tube, which allows the air to escape. The distention of the stomach with air pushes the apex of the heart upward and to the left. This pressure on the ventricles rotates the heart on its axis and distorts the great vessels at its base. The distended stomach or esophagus may disturb the heart in a reflex manner because the common innervation of these organs is through the vagi. Stimulation of the vagi causes a slowing of the heart. These factors often produce cardiac arrhythmias which may cause the patient considerable worry and anxiety.

Diagnosis.—The diagnosis of this condition is not difficult when the physician has an opportunity to observe the patient during a spell of eructation. The presence of food decomposition in the stomach should be ruled out by examination of gastric contents removed by the tube. By close observation the physician will notice that the patient collects a little saliva in his mouth, slightly flexes the head on the thorax, closes his mouth, and swallows. By this procedure the air is forced into the esophagus, producing a sound which leads the patient to believe that the act is an eructation, while just the opposite is the case. On opening the mouth the air is noisily belched, and then swallowing and eructation follow closely upon each other almost continuously. At times it requires eight or ten swallowings of air to induce one good eructation.

Treatment.—The treatment of this condition is largely psychic, and the physician must impress upon the patient the fact that he can prevent the condition himself if he will. The nature of the affection should be carefully explained to the patient, and he should be prevailed upon to cease the eructation as well as the frequent swallowing movements. The French recommend that the patient take a cork between his teeth to keep the mouth open; this is done after every meal, and the practice continued for a considerable length of time. While the mouth is open the patient cannot swallow air, and the eructations quickly cease. The patient can be instructed to wear a tight collar so that the pain of swallowing will attract his attention to the act. Then again, in order to keep the thyroid cartilage from rising, one may tie a ribbon moderately tight around the neck; this is not only a direct restraint but serves as a reminder to the patient.

Hyperalimentation is known to have a salutary effect upon weakened patients. Methodic treatment by sounds introduced into the esophagus is sometimes followed by beneficial results. The underlying nervous condition will in many cases yield to electricity,

change of climate, or hydrotherapeutics. The medicinal agents indicated in this condition consist of the bromids, belladonna, chloroform water, and preparations of valerian.

PNEUMATOSIS (DRUM-BELLY).

This term is used to designate a condition in which the stomach is greatly distended by air. The patient experiences symptoms which are referable to the heart, such as irregularity in rhythm, and dyspnea, as well as abdominal tension. Pneumatosis of a purely nervous character is due to the habit of swallowing air. Sometimes the condition is associated with simultaneous spasmodic closure of the pylorus and cardia, which renders it impossible for the air to escape. The distressing symptoms usually vanish with the expulsion of the air.

Treatment.—Pneumatosis is treated as are nervous eructations. The treatment should be directed toward increasing the strength of the organism as a whole. The drug treatment consists in the administration of bromids, cocain, and morphin, the latter either orally or hypodermically. The stomach tube will give immediate relief by allowing the air to escape.

NERVOUS VOMITING.

Nervous vomiting produced by disturbances of the nervous system, both central and peripheral, without external irritation or anatomic lesion, is a purely functional disorder. It occurs without any overexertion and is independent of the quantity and quality of the food ingested. It varies in relation to the different kinds of diet; is often absent when articles difficult of digestion have been eaten, and may be present when only suitable food has been taken.

Organic diseases of the central nervous system are not infrequently accompanied by vomiting of this nature. The gastric crises of tabetic patients are of peculiar interest in connection with this subject. They occur as a very early symptom of locomotor ataxia, and consist of violent attacks of vomiting, usually accompanied by intense gastric pain (*gastric crises*). The vomiting may last for days, placing the patient in a very grave condition. There are also purely motor gastric crises, which run their course without any sensation of pain, vomiting being the only distressing symptom. This latter condition is not responsive to treatment, which should be directed against the cause rather than the symptom. The cause, in a large majority of cases, is syphilis.

A few writers have described attacks of what they term idiopathic vomiting, which resembled very closely the gastric crises, and in which they were unable to detect any pathologic condition

of the spinal cord. Nervous vomiting is also frequently found in hysterical patients and in neurasthenics; it adds sometimes to the distressing symptoms of patients suffering from enteroptosis, atony, and nervous dyspepsia. Organic diseases, however, must be excluded before a diagnosis can be established. Nervous vomiting is very characteristic. It takes place with seeming ease, without preceding nausea; it is likewise independent of the quality of the food, but largely influenced by psychic causes. The general nutrition is, as a rule, easily maintained.

Treatment.—The treatment of vomiting of purely nervous origin is identical with that of neurasthenia, hysteria, and enteroptosis. In the presence of obstinate vomiting, recourse may be had to drug treatment, when such sedatives as cocain, menthol, morphin, chloral hydrate, valerian, validol, menthol-valerian, chloroform on ice, orthoform, or anesthesin may be used (see page 270).

Bismuth is one of the best drugs for the treatment of that class of vomiting which results from gastric irritation (see page 265). Cerium oxalate has probably the same action in allaying vomiting as bismuth. It has acquired a reputation in the treatment of the vomiting of pregnancy which clinical experience, as a rule, fails to confirm. Creosote, iodine and phenol may be grouped together as a series of drugs which allay vomiting that is produced by fermentative action in the stomach. The vomiting ceases upon removal of the cause. Hydrocyanic acid in small doses is another drug with a reputation in gastric vomiting; if, however, results are not obtained immediately, it is useless to persist with it. Aconite, in rather large doses, allays vomiting by inhibiting the reflex centers and thereby acting as a powerful sedative to the peripheral nerves in the gastric mucous membrane. It is one of the host of drugs suggested for the vomiting of pregnancy. Chloretone in doses of 0.3 to 0.5 Gm. (5 to 8 grains) relieves pain and often allays vomiting. For relief of the vomiting and pain of gastric crises, coryfin in 10-drop doses every two hours is often of great service. When vomiting is of reflex origin it is worth while to persist with potassium bromid, which may be given per rectum if not tolerated by the stomach. Vomiting in sea-sickness has been quickly relieved by inhibiting the vagus by the hypodermic injection of one or two doses of atropin 0.001 Gm. ($\frac{1}{80}$ grain). Chloretone in doses of 0.3 to 0.7 Gm. (5 to 10 grains), given by mouth, has acquired considerable reputation as a preventive of sea-sickness.

Hyperemesis or pernicious vomiting in pregnancy has yielded to treatment with epinephrin. Ten drops of a 1:1000 solution were given every morning and night, at first in an enema of 150 Cc. (5 ounces) of water, with 20 drops of tincture of opium, and after three days in ice-water by mouth. Curtis has had success in an obstinate case by the injection of blood from a normal pregnant woman. He injected 10 Cc. (5 iiss) of defibrinated blood into the

muscles of the back every two days. Recovery took place after three injections. Transfusing the patient with the blood of a normal postpartum woman will often relieve the exhausting vomiting and enable the patient to go over to full term. Good results are also reported from the hypodermic administration of soluble extract of corpus luteum, 20 milligrams ($\frac{1}{2}$ grain) in 1 Cc. (16 minims) of saline solution, the dose being given twice daily if necessary. Garnett believes that pregnant women develop an antigen which protects them from the toxins incident to the progress of pregnancy. Patients who are attacked with pernicious vomiting have failed to develop the specific antigen.

Persistent nervous vomiting will often produce acute irritation of the stomach. The important point is to get the irritated stomach to retain food. Even the retention of a liquid may break the vicious circle. If a single feeding can be kept down, the stomach will soon be able to stand another and more nutritious one. Sometimes a tablespoonful of brandy poured over another tablespoonful of cracked ice will act wonderfully well.

Opium may be administered in the form of suppositories. Morphine hypodermically administered acts as a powerful sedative to the vomiting center, and will afford relief in persistent and exhausting hyperemesis as definitely as it does in a paroxysm of pain.

Pressure or percussion of the fifth dorsal vertebra for one-half minute will often relieve the vomiting of pregnancy. This manipulation acts on the pyloric reflex and opens the pylorus (see page 211). The patient drinks a glass of water with 1 Gm. (15 grains) of sodium bicarbonate. Any member of the household is taught to strike a series of moderate blows on the fifth dorsal vertebra, which has been definitely located and marked. This manipulation is equivalent to duodenal lavage. After resting a few minutes until the nausea is abated, nourishment is given and the vertebra is again percussed.

Suggestive or psychic therapeutics and the use of gastric lavage, simple sounding and intraventricular galvanization have all produced favorable results with hysterical patients. It must not be forgotten that nervous vomiting is sometimes induced reflexly by a pathologic condition of other organs.

RUMINATION (MERYCISM).

Rumination is an unhappy faculty possessed by some patients by which they can at will bring back the food from the stomach to the mouth some time after it has been swallowed, to be again swallowed or expectorated. It is more common in males than in females. It affects neurasthenics, hysterical and epileptic persons, and sometimes idiots. In this class of patients rumination sometimes results from fright, rapid eating, overfilling of the stomach,

traumatism, or irritation of the stomach by chemical or thermic agents. It has been observed to develop in other patients by mere imitation; children of parents who ruminate are likely to indulge in the pernicious practice. The exciting causes mentioned induce, reflexly, anti- or retro-peristaltic movement, which results in the opening of the cardiac orifice, permitting the food to regurgitate to the mouth.

Rumination is frequently preceded by nervous dyspeptic symptoms of a mild nature, which become gradually aggravated until the fluid contents of the stomach are regurgitated. The voluntary regurgitation of food is not accompanied by nausea, and in many cases produces no discomfort whatever. In other cases, however, the food, having remained for a considerable length of time in the stomach, has become sour and disagreeable to the taste when regurgitated; the patients, annoyed, naturally spit it out. As might be expected, the habitual expectoration of food masses leads to marked emaciation of the patient. In these cases the secretion of gastric juice may show great variation from the normal, or it may be perfectly normal.

Treatment.—Psychotherapeutics must be resorted to as the chief factor in the treatment of these cases. The patient must be energetically persuaded to suppress the regurgitation of food. The nervous condition underlying the pernicious habit requires appropriate treatment. As a prophylactic measure, patients should be instructed to eat slowly and to thoroughly masticate their food. Such patients should not be left alone, either during the meal or for some little time afterward, since the presence of company imposes a salutary restraint on the ruminating habit. When the desire to ruminate arises, expiration of air should be postponed for a moment or two and swallowing movements suppressed. Patients should not talk while eating. It is important that defective teeth be either repaired or extracted. Children should be kept away from ruminants in order to avoid contracting the habit by imitation. Good results have followed the administration of acids in achylia, and large doses of alkalis in hyperacidity. Sometimes the patient experiences pain of greater or less severity in the region of the stomach when he attempts to suppress the practice of rumination, and in such cases warm applications or suppositories in which narcotic drugs are incorporated assist in relieving the distress. The bromids and strychnin are also indicated. The chief requirement in the treatment of this form of gastric neurosis is to fortify the will-power of the patient sufficiently to suppress the practice.

REGURGITATION.

Regurgitation proper is a condition in which the food returns involuntarily from the stomach to the mouth and is expectorated.

It may occur in health, but becomes pathologic when it persists over a prolonged period and when the quantity of food brought up is large. Emaciation results when patients regurgitate any considerable portion of the food ingested. The treatment of this condition is similar to that of rumination.

INSUFFICIENCY OF THE PYLORUS.

Pyloric insufficiency is a condition which has been known frequently to follow organic diseases. It has been noted after destruction of the pyloric sphincter by carcinoma or by pyloroplasty; cicatrices from gastric ulcer in the region of the pylorus; duodenal stenosis; catarrh of the stomach; and achylia. Pyloric insufficiency from purely neurotic causes is of exceedingly rare occurrence. Among the most important diagnostic indications of pyloric insufficiency is the fact that air blown into the stomach escapes immediately into the gut, thus rendering artificial distention of the stomach impossible. The flow of bile and of the contents of the small intestine into the stomach is likewise suggestive of a relaxed pyloric orifice. The diagnosis is easily made by means of the Roentgen ray. The degree of insufficiency is ascertained by the administration of the Ewald-Boas test breakfast (see page 96).

Treatment.—The treatment of pyloric insufficiency depends upon the cause of the disease. The clinician should endeavor to ascertain if there is any gastric secretion, and how soon after the ingestion of food the stomach becomes empty. The stomach contents should be aspirated one hour after the test meal is taken. If nothing be forthcoming, the test meal should be repeated and the stomach tube used at quarter-hour intervals after the meal. By this means it is possible to ascertain the quantity of gastric contents present at any time. The drugs indicated in this disease are such as aid intestinal digestion, since derangement of intestinal digestion, accompanied by distressing symptoms, is apt to arise from the premature passage of the food into the duodenum. The combinations of sodium and magnesium, rhubarb, ammonium chlorid, pancreatin, and bile in the form of inspissated ox-gall, are all useful. In diarrhea associated with this condition, strychnin has been found to give the best results. This drug should be rapidly pushed to the point of effectiveness. Many cases of diarrhea which have persisted for years have been known to respond most satisfactorily to the administration of strychnin sulphate in doses of 3 to 10 milligrams ($\frac{1}{20}$ to $\frac{1}{8}$ grain). The stools at once diminish in number and gain in consistency. Strychnin has been known to produce excellent results in about three and a half weeks in diarrhea resulting from insufficiency of the pylorus (see page 677).

SINGULTUS GASTRICUS.

Hiccup is a symptom manifested as a noise made by the sudden and involuntary contraction of the diaphragm and the simultaneous contraction of the glottis which arrests the rising air in the trachea. Singultus may last for a few minutes or much longer, or it may recur for days or months. It is a symptom often found in diseases of the abdominal viscera, such as gastritis, motor insufficiency of the first and second degrees, gastric carcinoma, enteritis, intestinal obstruction, appendicitis, cholera, pancreatitis (suppurative), diseases of the liver, and peritonitis; it has also been observed in the course of such diseases of the nervous system as epilepsy, tumor of the brain, meningitis, hydrocephalus, and hysteria.

In rare cases of singultus gastricus a continuous hiccup lasting for a long time, varying from weeks to months, and without regurgitation of food, may be present; there is usually, however, a hyperesthesia of the glandular layer of the stomach. Well nourished young adults, mostly young women, are the commonest victims of singultus. Occasionally it is a prominent symptom of gall-bladder disease and may be so incessant as to cause alarming exhaustion.

Treatment.—The treatment of the underlying cause is of great importance. For the symptom itself, citric acid and sodium bicarbonate, one teaspoonful of each, may be given separately; the resulting carbon dioxid distends the stomach so that it exerts pressure upon the diaphragm, relieving the spasm. Again, the patient assuming the dorsal decubitus, both thighs and knees flexed against the abdomen at the sharpest possible angle and pressed upward with force for a sufficiently long time, will cause the intestine to press against the diaphragm; the object is to remove the localized spasm by extension of the contracted muscle. Continued energetic pressure along the entire vertebral column frequently gives relief. External applications, such as a mustard plaster applied to the epigastrium, or a mustard paper to the back of the neck, are of great benefit. An enema containing turpentine will remove the gaseous distention of the abdomen. When the nervous element predominates, spirits of camphor or compound spirits of ether are advantageous. Chloral in doses of 1 Gm. (15 grains), repeated every two hours, controls the convulsive action of the muscles. Spasmodic irritability can be relieved by gelsemium, which depresses the respiratory center by diminishing the hypersensitiveness of the nerve centers; the fluid extract may be given in doses of 0.1 to 0.2 Cc. (2 to 3 minims), to be repeated every three or four hours. Quick relief is often brought about by 0.7 to 1.3 Gm. (10 to 20 grains) of chloretone. Ten drops of a saturated alcoholic solution of menthol in a little water, repeated every hour if necessary, may give relief. Some authors advise oil of amber in the dose of 1 Cc.

(15 drops) every two hours. Ten-drop doses of 1:1000 solution of epinephrin repeated in an hour will often prove efficacious. It may be necessary to check the spasmodic contraction of the diaphragm by inducing partial or complete general anesthesia. In some cases a hypodermic of morphin and atropin may be necessary. Continuous traction of the tongue will often bring quick relief. The hiccough can frequently be stopped by compressing the eyeballs as for the oculocardiac reflex (see page 390). This compression slows the pulse and induces sleep; it should be frequently repeated. The accompanying constipation should always have attention, and its cause must be determined. This is essential, as hiccough may be brought about by intestinal toxemia.

Recently benzyl benzoate has been found to be a useful remedy for hiccough. A 20-per-cent. solution in alcohol is prescribed, and of this thirty drops in water or milk every four hours. Benzyl benzoate is also of diagnostic interest in differentiating between hiccoughs of purely central origin and those which are due to some peripheral disturbance. Inasmuch as benzyl benzoate exerts its chief effect peripherally on the smooth muscle structures, it is most useful in the treatment of hiccoughs of peripheral origin (see page 276).

CHAPTER XVIII.

SENSORY NEUROSES.

GASTRALGIA; HYPERESTHESIA; GASTRALGOKENOSIS; NAUSEA;
BULIMIA; AKORIA; ANOREXIA.

GASTRALGIA.

GASTRALGIA, known also as cardialgia, gastrodynia, and neuralgia of the stomach, is a condition peculiar to individuals of a nervous temperament. The diagnosis cannot be confirmed until a careful exclusion is made of organic diseases of the stomach. The pains complained of in gastralgia are due to morbid or irritating conditions of the sympathetic nerve ganglia located in front of the spinal column. The site of the pain is the epigastric distribution of the lumbar sympathetic. The celiac plexus, the superior mesenteric plexus and the aortic plexus may also be involved. The location of the pain is in reality exterior to the stomach. The nervous gastric pains occur periodically and spasmodically, and at times become so intense as to be unbearable. The attacks last from a few hours to several days; the pains radiate toward the back and also up into the chest; they are usually independent of the reception of food. Nervous excitement is apt to bring on the attacks, during which vomiting rarely takes place. Eructations are common. The celiac plexus is often markedly sensitive to pressure exerted in the median line of the epigastric region. The superior mesenteric plexus, as well as the aortic, occasionally becomes very sensitive, as shown by pressure on two points situated immediately above and below the umbilicus. There is often found a hyperesthetic zone in the epigastrium. The differential diagnosis between gastralgia and ulcer of the stomach is fraught with difficulty, and established only after careful consideration of the symptoms of ulcer, such as pressure points, relation of the pains to the reception of food, and occult hemorrhage. In ulcer the hyperesthetic cutaneous zone is usually smaller in area than in gastralgia. Among the recognized causes of gastralgia are syphilis, gallstones, and chronic appendicitis; the pain is reflex, through the sympathetic system.

Treatment.—The treatment of gastralgia should be directed toward the generally debilitated condition of the patient, and should embrace, among other things, hydrotherapy, change of climate, and the milk cure. Very little or no restriction need be made in regard to diet, since the condition is purely extragastric. It

is not necessary that patients should be kept on either fluid or light diet. The regimen may be varied and generous in quantity without aggravating in any way the painful symptoms. The diet, however, should be suited to the individual case. It will be necessary in many cases of this class to persuade patients to eat, and to impress upon them that there is no connection between the ingestion of food and the pains of which they complain, but that there is danger of aggravating the symptoms by abstaining from food.

During the acute attack the patient should be put to bed and hot compresses or poultices should be applied to the region of the stomach. Good results are obtained by a "half-bath" or a protracted hot sitz bath (see Chapter XII). When the pains are of a violent nature, resort must be had to such drugs as morphin or opium and belladonna in combination.

	Gm. or Cc.	
R—Morphine sulphatis	0 01	gr. 1
Extracti belladonnæ	0 02	gr. 1
Olei theobromatis	2 0	gr. xxx
Misce et ft. suppos. no. 1.		

Sig.—As required for the relief of pain.

	Gm. or Cc.	
R—Extracti opii	0 05	gr. 1
Extracti belladonnæ	0 02	gr. 1
Olei theobromatis	2 0	gr. xxx
Misce et ft. suppos. no. 1.		

Sig.—As required for the relief of pain.

	Gm. or Cc.	
R—Cocainæ hydrochloridi	0 5	gr. viiiss
Aquæ auranti	30 0	5j
Aquæ chloroformi	75 0	3iiss
Aquæ destillatæ	45 0	5iss

Misce.

Sig.—One to three teaspoonfuls in water at the beginning of the attack.

In severe cases morphin should be given hypodermically at once. Among the medicaments which may be administered by mouth are cocain (0.05 Gm. to 150 Cc. of water, in teaspoonful doses), codein phosphate (0.03 to 0.05 Gm.— $\frac{1}{4}$ to 1 grain), chloral hydrate, antipyrin (0.5 Gm.), acetylsalicylic acid (1 Gm.), chloroform water, validol, and the ammoniated tincture of valerian. Extract of cannabis indica has also been recommended for relief of the pains.

	Gm. or Cc.	
R—Extracti cannabis indicæ	0 03	gr. ss
Sacchari albi	0 50	gr. viij
Misce et ft. pulv. no. 1.		

Sig.—One every four hours.

	Gm. or Cc.	
R—Tincturæ cannabis indicæ	4 0	3j
Tincturæ valerianæ	6 0	3iiss

Misce.

Sig.—Twenty drops to be taken at a dose.

Hoffman's anodyne, 20 to 30 drops on a lump of sugar, is valuable in the treatment of this condition. Hot drinks such as peppermint tea or valerian tea are productive of favorable results. If convenient, galvanization is worthy of a trial, when the anode should be placed over the epigastrium and the cathode over the spinal column for five to ten minutes. The faradic current may also be used. In cases where pains are less violent but of prolonged duration, massage and electricity are indicated in addition to warm applications.

A special form of gastralgia is represented by the gastric crises of locomotor ataxia, which are characterized by violent cramps in the stomach and pains in the back, followed by vomiting. As to the nature of these crises we are still in the dark, nor have we by any means been able to cut short the attacks except by the use of morphin or the injection of cocain to anesthetize the posterior roots. It is an advantage to distinguish between vagus and sympathetic gastric crises. The vagus is involved when there is pain, vomiting, tachycardia, and disturbance of the larynx. Sympathetic crises are more frequent. König recommends injecting 100 Cc. (3 ounces) of a 0.5-per-cent. solution of novocain into the muscles of the back on a line each side of the spinous processes. Alcohol can be injected in the same way; the results are similar to those obtained by its use in the treatment of facial neuralgia. The following drugs are recommended in addition to the measures already mentioned in connection with the treatment of the acute painful seizures: Antipyrin 0.6 Gm. (10 grains), or cerium oxalate 0.3 to 0.6 Gm. (5 to 10 grains), three times a day; acetylsalicylic acid and the salicylates.

	Gm. or Cc.	
R—Sodii salicylatis	8 0	3ij
Caffeine sodiosalicylatis	2 0	gr xxx
Aque q. s. ad	50 0	3ij
Misce.		

Sig. — 1 to 2 Cc. (15 to 30 minims) of the sterilized solution to be injected daily into the median vein. (Von Mendel.)

In the operation of rhizotomy Foerster says the aim is to resect the sensory gastro-intestinal fibers of the sympathetic nerve, and this may require resection of the roots from the twelfth to the fifth dorsal or even higher. It may be possible to determine beforehand exactly the roots requiring resection, by careful study of the location of the pains and of the superficial hyperesthetic zones. There is always a possibility that the crises may be due to the vagus or to direct irritation of the vomiting center in the medulla oblongata, in which case rhizotomy of course would not relieve. If the blood supply of the spinal cord is interfered with, paralysis may result. Many of the patients after the operation of rhizotomy have been restored to comparative health and enabled to return to business. Franke's operation has been employed with good effect in the

treatment of tabetic gastric crises. The aim is to pull out the intercostal nerves and thus realize by a comparatively simple technic results equivalent to those of resection of the posterior spinal nerve roots, namely, interrupting the continuity of the fibers of the sympathetic nerve innervating the seat of the pains. The reflex arcs have also been broken by severing the vagus. Vagotomy (resection of the vagus) frequently frees the patient from gastric crises (see page 783).

The majority of gastralgias are secondary affections and may occur in the course of almost any affection of the stomach, intestine or other abdominal organ, in arteriosclerosis, toxemias, anemia, and diseases of the male and female sexual organs. In all such cases treatment should be directed toward the primary cause.

GASTRIC HYPERESTHESIA.

Gastric hyperesthesia is defined as an increased sensitiveness of the gastric mucous membrane to chemical, mechanical and thermic stimuli, or to any one of these. A patient with a good appetite may suffer pain when certain articles of food or drink are taken, which is not relieved until the food or drink has disappeared from the stomach. The stomach is often hypersensitive to sugar, fat, and carbohydrates. Of thermic stimuli the stomach is more sensitive to cold than to heat. The abnormal sensations may vary, amounting in some cases to severe pain and vomiting. During digestion there may be sensations of fulness, pressure, tension, or burning, but these usually cease with the evacuation of the stomach. Gastric hyperesthesia is a condition rather frequent in neurasthenic and hysterical subjects. Patients come to associate the distressful symptoms with the ingestion of food, and as a result the quantity of food consumed becomes less and less and the patient loses flesh and strength.

Treatment.—The treatment should be directed against the cause. Efforts should be made to improve the general nutrition, and, if necessary, a course of hyperalimentation (see page 569) should be instituted. Asthenic patients require rest in bed, and should be kept absolutely quiet both mentally and physically. The dietetic cure should be commenced with caution; it should consist at first of milk and kefir, to be gradually changed to a diet of semisolid consistency. The dietary should be such as to reaccustom the patient to ordinary food. When the distaste for food is very marked it may be necessary, at times, to resort to nutrient enemata (see page 243).

Nitrate of silver is particularly effective in diminishing the sensitive condition of the stomach. A tablespoonful of a solution of 0.2 to 0.3 Gm. (3 to 5 grains) to 100 Cc. (3iiss) of water is given three times a day. It is best administered as a tablespoonful

of the solution to a wineglass of distilled water, before breakfast, and half an hour before dinner and supper. Lavage with silver nitrate solution (1:10,000) also acts well. When there is much pain, belladonna, chloroform or a preparation of valerian is indicated. Anesthesia may be given in doses of 0.25 to 0.5 Gm. (4 to 7½ grains), ten to fifteen minutes before meals. For the relief of gastric irritation Rochester advises the following combination:

	Gm. or Gs.	
R—Strontii bromidi	6.0	3iss
Sodii bicarbonatis	40.0	3x
Carbonis ligni	20.0	3v
Bismuthi subcarbonatis	20.0	3v
Magnesiæ	180.0	3vj

Misce.

Sig. Two teaspoonfuls in water three times a day, after meals.

If between meals patients are troubled with burning or pain in the stomach which may be accounted for by the presence of hyperacidity, Stockton's sedatives will be found satisfactory.

When the gastric distress is severe and hyperacidity exists:

	Gm. or Gs.	
R—Ceri oxalatis	1.0	gr. xv
Bismuthi subcarbonatis	2.0	3ss
Magnesiæ carbonatis	4.0	3j

Misce

Sig. +A teaspoonful stirred into one-half glass of water. (Stockton.)

When the action of the magnesia is too laxative, and for this reason we desire a gastric sedative that will quiet the bowels:

	Gm. or Gs.	
R—Ceri oxalatis	1.0	gr. xv
Bismuthi subcarbonatis	4.0	3j
Cretæ præparata	4.0	3j
Carbonis ligni	2.0	3ss

Misce

Sig. -Teaspoonful well stirred into one-half glass of water. (Stockton.)

GASTRALGOKENOSIS.

The term "gastralgokenosis" is used to designate stomach-ache or the sensation of painful pressure in the region of the stomach when that viscus is empty. In this condition there is hyperesthesia of the empty stomach. The pain may become very severe a few hours after eating, when the stomach is empty. Excess of hunger has never been observed to accompany this condition, though pain is promptly relieved by the ingestion of food. Duodenal ulcer must be differentiated (see Chapter XLI).

The patient should endeavor to ward off the attack by never permitting the stomach to become quite empty. He should have with him always some articles of food, such as crackers or milk. Small doses of extract of opium, 0.006 to 0.008 Gm. ($\frac{1}{16}$ to $\frac{1}{8}$ grain), and the bromids, are indicated for this condition.

NERVOUS NAUSEA.

Idiopathic nausea appears most frequently in women and in consequence of a general neurotic condition, anemia and chlorosis, or disturbance of the menstrual function. Purely functional nausea may occur at intervals. Mental disturbance acts as an exciting cause. Nervous nausea may occur in the morning while the stomach is empty after the night's fast. During the attack, patients experience a pronounced aversion to food. The disease may at times assume an obstinate form, which may be due to the variable condition of the blood supply to the brain. The gastric functions, in the majority of cases, are normal; rarely a moderate degree of hyperacidity may be present.

Treatment.—When the nausea is due to neurasthenia or anemia, these conditions should receive attention. Anemia may be remedied; that is, hemoglobin can be rapidly increased by the hypodermic use of the citrate of iron, as described on page 581. Sometimes the hyperalimentation cure should be instituted in cases where the general nutrition is low. Patients occasionally do well when removed from their homes and customary surroundings. Particular attention should be paid to the mental state of the patient, which is often depressed. Severe cases should be treated in a properly conducted hospital or a sanitarium. Food should be served in an attractive manner, for the sake of its appetizing influence and the pleasure which details of this kind give the patient. Should there be nausea early in the morning, it will be well to serve breakfast in bed. Hydrotherapeutics will be found a valuable factor in the treatment. For cutting short the attack of nausea, a bath at 64° F. or a cold douche is recommended. Both intraventricular and extraventricular galvanization may be employed. The bromids, chloral (3 to 5 grains three or four times daily), and valdol (six to eight drops every two or three hours) are indicated in the treatment of nervous nausea.

BULIMIA.

Bulimia, cynorexia, and hyperorexia are terms used to designate a condition in which the sensation of hunger is more frequent and more intense than in the normal state. Bulimia may be a primary affection, or it may be associated with other diseases, as gastric ulcer or carcinoma, hyperacidity, pancreatic affections, exophthalmic goiter, hysteria or neurasthenia; and the condition may be either acute or chronic. "In the midst of perfect euphoria, a feeling of intense hunger overcomes the patient, with a desire to satisfy it. This hunger sensation is associated with a gnawing feeling in the stomach, and the utmost fear and anxiety, as if something alarming were going to happen. If the feeling of hunger

is not satisfied very quickly, severe headache and trembling of the body, or even fainting spells, may occur." (Einhorn.) The attack of bulimia sometimes yields to the ingestion of a small amount of food, but as a rule large quantities have to be taken.

Treatment. The cause should be carefully ascertained. Simple articles of food or drink, such as crackers, zwieback, chocolate, or milk, should be carried by those who are subject to these attacks, or be at all times within convenient reach.

Efforts have been made to influence the irritable condition of the "hunger center" by the use of drugs. Bromids in large doses, 1.5 to 2 Gm. (25 to 30 grains), may be given two or three times daily. Opium with belladonna can be given, as follows:

	Gm. or G ^{ss}	
R ^x —Extracti opii,		
Extracti belladonnæ	āā 0 01	gr 1
Misce et ft. caps. no. i.		
Sig.—One capsule to be taken morning and night.		

Arsenous acid, 0.001 Gm. ($\frac{1}{100}$ grain), in pill form, or liquor potassi arsenitis, may be administered. Cocain may be prescribed in the following form:

	Gm. or G ^{ss}	
R ^x —Cocainæ hydrochloridi	0 1	gr 100
Aquæ amygdalæ amaræ	10 0	3 iiss
Misce.		
Sig.—Ten drops several times a day.		

Or:

	Gm. or G ^{ss}	
R ^x —Ammonii bromidi,		
Sodii bromidi	āā 8 0	3 ij
Aquæ menthæ piperitæ	60 0	3 ij
Misce.		
Sig.—One teaspoonful twice daily.		

AKORIA.

In patients suffering from akoria the normal sensation of satiety is lacking, even after a full meal. Patients do not know when they have eaten enough. There may be no particular desire for food, however, and even well-marked anorexia may be present. Akoria is found in connection with such conditions as give rise to bulimia or polyphagia. Neurasthenic and hysteric subjects are among its victims. The treatment should consist of change of climate, hyperalimentation, hydrotherapy, electricity, and psychotherapeutics.

NERVOUS ANOREXIA.

Nervous anorexia is a term used to designate loss of appetite of a pronounced and chronic nature. There exists on the part of the patient a repugnance to every kind of food. In spite of this

fact, the functioning powers of the stomach and intestine are, as a rule, normal. The disease is apparently characterized by anesthesia of the hunger "nerves." Nervous anorexia is always a symptom of such general nervous conditions as neurasthenia, sexual neurasthenia, and hysteria. It may result seriously, from lack of proper nourishment to the body. Among the exciting causes are frequently found great mental depression, worry, anxiety, and fright.

Treatment.—This consists in maintaining the nutrition, if need be, by means of the so-called food or hyperalimentation cures. Removal of the patient from his home surroundings must be considered in grave cases. Sometimes it is necessary to resort to nutrient enemata or to gavage. In gavage, or feeding by the stomach tube, such nourishment as milk, eggs, gruel or artificial food is poured into the stomach through a funnel that fits into the external end of the tube. Care must be exercised not to cause too much distention of the stomach, unaccustomed to food in even ordinary quantities, until tolerance has been established.

Stomachics, such as orexin, 0.3 Gm. (5 grains), three times a day, two hours before meals, and cinchona bark preparations, are indicated.

	Gm. or Co.	
R—Decocti cinchonæ	10-180 0	℥iiss-℥vj
Acidi sulphurici diluti	0 3	℥v
Syrupi zingiberis q. s. ad	200 0	℥vj
Misce.		

Sig.—One tablespoonful three times a day, half an hour before meals.

	Gm. or Co.	
R—Fluidextracti cinchonæ	60 0	℥ij

Sig.—One-half teaspoonful in a wineglass of water, to be taken three times a day (before meals).

The stomach may also be washed out with water in which stomachics have been incorporated. Arsenic, iron, small doses of the bromids, and strychnin (0.001 to 0.002 Gm., $\frac{1}{80}$ to $\frac{1}{40}$ grain) may be prescribed as occasion requires.

CHAPTER XIX.

NERVOUS DYSPESIA NEURASTHENIA GASTRICA.

VON LEUBE, who first described the condition designated nervous dyspepsia, included those subjective nervous symptoms which are, as a rule, of marked intensity, and for which it was impossible to find cause in any of the organic or functional disturbaners of the stomach. Later, however, it was discovered that nervous symptoms of a like nature present themselves in disturbances of the sensory, secretory, and motor functions of the stomach. As a consequence the term *nervous dyspepsia* as now employed signifies a gastric neurosis which is entirely independent of organic disturbances, being of purely nervous origin, although there may be coincident disturbances of the motor, sensory, or secretory functions.

Etiology. The cause of nervous dyspepsia must be sought in the increased irritability of the autonomic nervous system. This heightened irritability, however, shows itself but rarely as an independent affection. The class of persons apt to be sufferers from this condition are possessed of a more or less irritable and unstable central nervous system. They are spoken of as being of a *nervous disposition*. In such persons fright, sorrow, care, pathophobia, or other emotion, often acts as an exciting cause of actual dyspeptic conditions.

Gastric Neuroses and Eye-strain. The medical profession is indebted to Dr. George M. Gould for the persistency with which he has maintained that many of the so-called gastric neuroses are due to eye-strain. Nervous dyspepsia resulting from eye-strain is characterized by such symptoms as sick headache, anorexia, anemia, and many types of malnutrition, all of which may be due to astigmatism or anisometropia. The influence of the visual organs over the digestive system may be proved by the fact that a normal person wearing glasses that may be worn with comfort by another becomes nauseated, even to the point of vomiting. There is no truth in medical science more susceptible of demonstration and more persistently ignored in daily practice than the immediate association of eye-strain and malassimilation. Headaches of all kinds, sick headaches, migraine, hemicrania, "rush of blood to the head," are usually due to eye-strain. My personal experience confirms the belief that eye-strain is frequently the main etiologic factor in nervous dyspepsia, since beneficial results are obtained as soon as the errors of refraction are properly corrected.

Cholecystitis, cholelithiasis, appendicitis, constipation, enteroptosis, or helminthiasis may induce nervous dyspepsia, probably by transmission of the irritation of the intestinal sympathetic nerves to the nerves of the stomach. *Epigastric hernia* is frequently a cause of this neurosis; as are also diseases of the female reproductive organs, such as *salpingitis, lacerated cervix, lacerated perineum, and anomalies of menstruation*. During the menstrual period the gastric secretion is often hyperacid. In the male, *sexual excesses* are not infrequently responsible for nervous dyspepsia. *Hemorrhoids* are a very common cause. Among the etiologic factors must also be mentioned diseases of such remote organs as the lungs, heart, and kidneys.

It has long been recognized that patients who for years had been treated for nervous dyspepsia have been cured after an acute attack of *appendicitis* which necessitated operation. The condition of the appendix had not been considered in connection with the treatment as the cause of the nervous dyspepsia. Unsuspected gallstones may often produce symptoms of nervous dyspepsia. A sudden attack of gallstone colic draws our attention to the gall bladder; and after removal of the offending gallstones the gastric symptoms entirely disappear.

There is a vast amount of suffering, manifested by general ill health, vague stomach and intestinal symptoms, progressive loss of flesh and strength, obscure nervous conditions, anemia, and obstinate constipation, occasioned by chronic appendicular inflammation. The pathologic condition in these cases is often due to a slow proliferation of connective tissue in the walls of the appendix. There is no pus formation, and it may be months or years before an attack of acute appendicitis occurs to clear up the diagnosis. When pain is felt in these cases, it is often not in the region of the appendix, over the McBurney point; it may be more generalized about the umbilicus or be referred to the site of the gall bladder or be felt in the stomach. These referred pains are not surprising when we remember the abundant connections of the rich nerve supply of the appendix, through the superior mesenteric plexus of the sympathetic, with the pneumogastric, hepatic, and gastric plexuses. These cases are diagnosticated as nervous dyspepsia and are treated expectantly and symptomatically; when the physician has exhausted his patience and resources he classes them among neurasthenics, and is likely to send them on a sea voyage, to a mineral spring, or to a sanatorium. Many such cases recover when the appendix or the gallstones are removed. Physicians should be on the alert for concealed appendicitis, well named by Ewald "*appendicitis larvata*." Obscure as it is, the true diagnosis will often be reached only by a process of exclusion (see page 772).

Symptoms.—Nervous dyspepsia gives rise to a variety of symptoms, some of which are general in character while others are

referable particularly to the stomach. It is at times a difficult matter to differentiate between symptoms of purely nervous origin and those which have an organic or functional basis. The variety and variability of the symptoms, as well as the manner in which the patient describes them, are characteristic of the condition present. Often such patients are free from distressing symptoms for days, or even weeks, when, owing to some trivial cause, most likely of a psychic nature, a recurrence of the symptoms takes place. The appetite of the patient is apt to be precarious: coarse food in large quantities may perhaps be partaken of without aggravation of the symptoms, while, on the other hand, certain dietetic articles which might be taken with impunity by a person suffering from organic disease of the stomach are rejected as "not agreeing" with the patient.

The general symptoms of which patients suffering from nervous dyspepsia complain are: fulness of the head, cephalalgia, migraine, inability to work, vertigo, lassitude, insomnia, hypochondriac and melancholic illusions. Opposed to this catalogue of subjective symptoms the objective symptoms are often inconsiderable. Patients, as a rule, exhibit the well-known neurasthenic type. The condition of nutrition is usually good. When the subjective symptoms become severe, there is at times a diminution in weight, owing to the refusal of the patient to partake of adequate nourishment. A condition of genuine inanition may develop. Palpation of the stomach often elicits hypersensibility to pressure over the celiac plexus. Cutaneous hyperesthesia is sometimes found over the region of the stomach. While all this is present the stomach may function in a perfectly normal manner. In other cases hyperacidity, subacidity or achylia may be present, either singly or in combination with atonic conditions of the stomach. It is a peculiarity of nervous dyspepsia that well-marked variability in the secretory functions sometimes exists, so that in the same patient achylia, subacidity, normal acidity, and hyperacidity may be discovered at different examinations—heterochylia (see page 94).

The physician should carefully examine the entire gastro-intestinal tract in every instance of suspected nervous dyspepsia in order to confirm or establish his diagnosis. Oftentimes an accurate diagnosis is only achieved after a prolonged period of observation.

Prognosis.—The prognosis for a complete cure without recurrence is not favorable. It is possible, however, under proper treatment, to bring about marked improvement in the condition of the patient.

Prophylaxis. As prophylaxis, the children of neurotic individuals in whom the habitus enteroptoticus is well marked should be kept well nourished and should be given gymnastic exercises. The avoidance of excessive mental exertion is an important prophylactic measure.

Treatment.—The treatment of nervous dyspepsia *per se* should be directed toward correcting the causes, whatever they may be. If the nervous dyspepsia is secondary, the primary condition should receive appropriate treatment. The subjective symptoms of the patient yield most readily when he is removed from his customary environment and is accorded complete rest of both body and mind. Marked benefit has resulted in some cases from a six weeks period of absolute rest. During the rest cure, so-called, the patient's mind should be occupied as little as possible. The relationship of the physician and patient in such cases is of the utmost importance. That physician will have the greatest success, other things being equal, who knows how to gain the confidence of his patient and is able to exert an influence over him. Many patients, however, owing to domestic or financial circumstances, are unable to leave their home surroundings. Such patients should be instructed to lie down for an hour or two, regularly, every morning and afternoon.

The question of nutrition is of paramount importance. The diet should be adapted to the individual case, and greater latitude may be permitted in regard to variety and quantity than in cases of organic gastric disease, since the dyspeptic symptoms are not intimately connected with the food in the stomach. An effort should be put forth to maintain the nutrition of the patient to the greatest possible extent. When constipation is present, coarser foods and foods leaving a considerable residue are indicated. Even when the general nutrition is normal a course of hyperalimentation may put an end to the nervous symptoms (see page 569). When, however, well-marked secretory disturbances are present, the diet should be adapted to the condition of the secretion. Atonic states of the stomach must be likewise considered in prescribing diet. Patients whose symptoms appear synchronously with the entrance of food into the stomach should receive a bland, non-irritating diet at the commencement of the treatment, to be gradually changed to one of a more solid consistency. When an aversion or distaste for meat exists, other protein foods must be substituted. Sometimes, however, a purely vegetarian diet is followed by good results. When meat is eliminated from the diet, there is a corresponding diminution of gastric secretion. To supply the needed stimulus to secretion, meat extracts may be prescribed. The meat-free lactovegetable diet, as sometimes prescribed in gastric diseases, is not identical with that of the strict vegetarian, who places great stress on the consumption of raw fruits and vegetables. A vegetarian diet proper would be too coarse, too voluminous, and too poor in iron, for patients with gastric disease. For the nervous dyspeptic the so-called lactovegetable diet is worthy of consideration; this diet includes certain animal products, such as milk, butter, cheese, and eggs. Coarse indigestible food

422 NERVOUS DYSPEPSIA—NEURASTHENIA GASTRICA

should be avoided by patients suffering from nervous dyspepsia, and the diet should possess as high a nutritive value as possible in proportion to the amount ingested. Such foods as radishes, celery, fresh fruit, nuts, almonds, dates, horseradish and mushrooms should not be permitted this class of patients. These food articles are, however, suitable for dyspeptic patients suffering from constipation, provided the state of their nutrition is good and their symptoms are not associated with the ingestion of food.

In prescribing a lactovegetable diet the physician should take into consideration the individual requirements of the patient. While green vegetables may be used in large quantities, they should be prepared and served in a finely divided state or in the form of purée. Dry vegetables and leguminous flours rich in protein should be prescribed in liberal quantities. Flour and egg dishes in the form of puddings, jam, and fruit juices are well borne by the nervous dyspeptic. The unfermented juice of grapes, possessing a comparatively high nutritive value, is a suitable beverage. Of baked foods, wheat bread, zwieback, rusks, biscuits, and brown bread may be prescribed (see page 655).

LACTOVEGETABLE DIET LIST (WEGELE).

		Protein	Fat.	Carbohydrate.
Morning	250 Gm. milk cocoa . . .	9.0	10.0	72.50
	100 Gm. rolls . . .	9.0	1.0	58.00
	30 Gm. butter . . .	0.5	24.6	0.15
Forenoon	250 Gm. milk pap with white of egg . . .	12.0	8.0	11.00
Noon	250 Gm. vegetables with rice	5.0	18.0	20.00
	250 Gm. pudding . . .	15.0	25.0	50.00
	150 Gm. apple-sauce . . .			20.00
Afternoon	250 Gm. milk cocoa . . .	3.0	10.0	72.50
	100 Gm. rolls . . .	9.0	1.0	58.00
	30 Gm. butter . . .	0.6	24.6	0.15
Evening	200 Gm. gruel with yolk of egg . . .	3.5	7.5	18.00
	200 Gm. water noodles . . .	5.0	1.5	40.00
	125 Gm. plums . . .	0.4		8.30
	100 Gm. rolls . . .	9.0	1.0	58.00
	30 Gm. butter . . .	0.6	24.6	0.15
		81.6	156.8	486.75
Calories		300.0	1300.0	2060.00

Total combustion value, 3600 calories.

The protein in lactovegetable diet may be supplied in the form of eggs, milk, and cheese; the fat constituent of such diet is derived from butter, oil, milk, and cream. Milk should be fed to this class of patients in large quantities, pure, or as buttermilk, sour milk, kefir, or yoghurt (see page 164). Yoghurt is said to possess the power of lessening putrefactive processes in the intestinal tract. In prescribing a lactovegetable diet the condition of the gastric secretion should be closely studied. In order to assist the patient

in maintaining a fair appetite, monotony in articles of food prescribed should be avoided. Of beverages, tea is preferable to coffee. Alcoholic drinks should be avoided entirely.

Physical Treatment.—Hydrotherapeutic measures are indicated for the general nervous condition which characterizes patients suffering from nervous dyspepsia. These measures consist of cool rubbings, half-baths, cool douches, and cold baths. The Scotch douche, alternate cold and hot applications, may be used locally over the region of the stomach. (See Chapter XII.) Patients whose state of nutrition is good should be persuaded to persevere in gymnastic exercises; those in a run-down condition should not undertake exertion of any kind before the condition of their nutrition has improved. Massage, including vibratory treatment, may be instituted for the purpose of stimulating tissue metamorphosis; it may include the whole body or simply the stomach or abdomen. Electric treatment, galvanic or faradic, of the stomach and intestine, may be applied with advantage. (See Chapter X.)

Whenever gastroenteroptosis is a complication in nervous dyspepsia, the treatment indicated for this condition and described in detail in Chapter XXX should be instituted.

Sea-water Therapy.—Good results have frequently been obtained from the subcutaneous injection of isotonic sea water in the treatment of cases of nervous dyspepsia of obscure origin. The results, if beneficial, are apparent soon after beginning the treatment. It has been clinically demonstrated that sea-water plasma is a powerful tonic to the nervous system. It stimulates metabolism to such a degree that the appetite improves and there is an increase in the body weight. The water relieves pain, allays nervous irritability, and induces restful sleep; there is a general improvement in tone throughout the entire nervous system, and the bowels move regularly.

The therapy of sea water depends upon Quinon's law of marine constancy: "Animal life, which appears as a cell in seas of well-determined saline concentration, in order to maintain its optimum cellular activity has always a tendency throughout the zoological scale to keep the cells of which each organism consists in the aquatic marine conditions of their origin."

Geology and paleontology agree in admitting that animal life first appeared in the sea, and the analysis of the blood serum and ash of every animal in the entire zoological series shows that the mineral composition of the medium necessary to cellular life is the same as that of the original seas. It is from these facts that Quinon deduces his novel conception of the animal organism as an actual aquarium in which the cells of which it is composed continue to live under the aquatic conditions of their origin.

Having shown that the primordial seas contained only 0.8 per cent. of salts, it is necessary, in order to produce a plasma of that

strength, to dilute the sea-water of the present day, which contains 3.3 per cent. For the purpose of diluting, pure spring water containing a minimum of mineral matter and free from bacteria is used in the proportion of five parts to two parts of sea water. Great care must be observed in collecting the sea water in order to avoid accidental impurities.

It should be obtained not less than twenty miles from any port or any stream flowing from a port, and at a depth of not less than ten meters. The water must be fresh, three weeks being the limit of time which should elapse between its collection and its injection. After dilution as above it should be filtered through a porcelain filter of the Pasteur type. Every precaution for the sterilization of vessels should be observed, but the water, aside from the care in handling and filtering as above, is not to be sterilized further, or it will be rendered therapeutically useless. After filtering the water it may be put in flasks or ampoules of a capacity of 30, 50, 100 Cc. or more, as the convenience of the operator may require.

The injection is performed with a rubber tube 1.5 meters in length and ending in a platinum-iridium needle 3 centimeters long; this latter should be protected by a glass tube (Fig. 81, *T*). The tube and needle must be boiled before connecting with the ampoule. The connection is made as follows: (1) File the lower end of the straight tube, *A*, of the ampoule, break its point and join it to *B*, the free end of the rubber tube. (2) File the end of the bent tube *C*, break its point, and hang up the ampoule by the bend in the tube at *D*, about one meter above the patient.

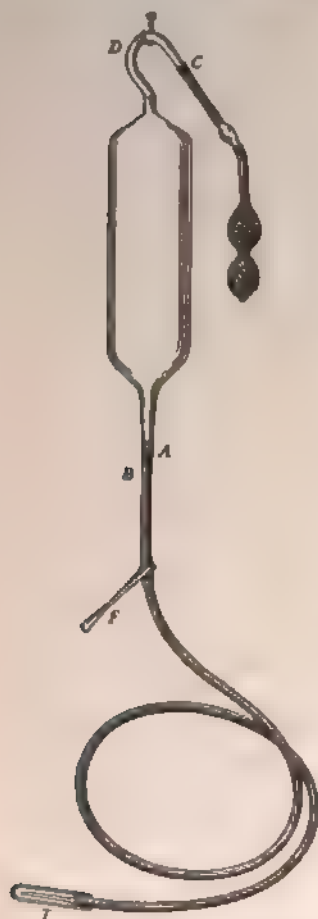


FIG. 81.—Apparatus for the injection of sea water.

To start the flow, the bulb of a thermocautery attached to the end *C* is useful. It is advisable to interrupt the tube of this bulb by a glass tube packed with sterilized absorbent cotton to filter the air. (3) The ampoule being hung up, remove the glass tube (*T*) and allow the fluid to run until the rubber tube is quite empty of the boiled water and the air it has contained. Make sure it is salt

water that is running by tasting drops on the back of the hand periodically. Then stop the flow with the clip *F*.

The best point for injection is behind the great trochanter. After the skin has been cleansed with alcohol the needle should be driven its whole length at right angles to the skin surface, except in very thin persons. If this should cause pain, withdraw the needle a few millimeters. Subsequent injections should be made in the same location, twelve hours after the first injection, to avoid a repetition of the pain which may arise from stretching of the tissues. After injection the needle wound should be covered for a minute or two with a pledget of cotton soaked in alcohol; it will have closed up by that time.

The quantity of sea water injected is of great importance. It is advisable to start with 20 to 25 Cc. (7 to 8 ounces). There should be no rise of temperature or other symptom of reaction. If symptoms of malaise appear, the dose must be decreased; if there is no reaction, it should be increased to 50 Cc. as soon as possible, to be administered every other day. The injections may be given at any time of the day. The treatment need not cease on account of menstruation. When it is well tolerated the dyspeptic symptoms diminish progressively, and recovery is brought about after a course of thirty to forty injections.

Drug Treatment.—Drugs occupy an important place in the treatment of this type of dyspepsia. The tonics, stomachics, sedatives and hypnotics are all valuable. When nervous irritability is marked, the bromids may be prescribed. Chloral hydrate in small doses, 0.1 to 0.3 Gm. ($1\frac{1}{2}$ to 5 grains), may be prescribed, to be taken three or four times a day. Insomnia may be combated by veronal, trional, or chloral hydrate, the last in the dose of 2 or 3 Gm. (30 to 45 grains) per rectum in a mucilaginous vehicle. Deficient appetite calls for bitters and stomachics.

When constipation is a complication it should be treated by other means than purgatives. A diet should be prescribed which leaves large residues in the intestine (see Chapter VII); abdominal massage, faradization of the rectum and abdomen, and enemata in which olive oil or cottonseed oil has been incorporated, will usually serve to counteract the constipation (see page 223).

Glycerophosphates and lecithin have been used with marked success in the treatment of nervous dyspepsia. In addition to the present official elixir containing the glycerophosphates of sodium and calcium, a compound elixir containing the glycerophosphates of calcium, sodium, iron, manganese, quinin and strychnin is largely used. The glycerophosphates and lecithin in various combinations are placed before the profession in ampoule form, and may be administered hypodermically, together with the iron and arsenic preparations described on page 351. Cacodylate of sodium hypodermically, 0.5 Gm. ($7\frac{1}{2}$ grains), has proved of great value in

my work. I give this preparation once a day for four weeks. It is a great stimulant to metabolism, and so affects nutrition as to bring about marked improvement in the general nervous condition.

Menthol has proved valuable in the treatment of nervous dyspepsia. The pain, vomiting, anorexia or flatulence often subsides at once, and permanent relief results. It should be prescribed in the dose of 0.3 Gm. (5 grains), three times a day. It can be advantageously combined with the alkalis.

The author has found the following prescriptions of value:

In cases of hypersecretion:

	Gm. or Co.	
R—Extracti belladonnæ foliorum	0 5	gr. viiss
Magnæs magnesiat q. s. ad	120 0	3iv
Misce.		

Sig.—Teaspoonful three times daily, a quarter of an hour before meals.

In cases of fermentation add resorcinol:

	Gm. or Co.	
R—Resorcinolis	5 0	3iiss
Extracti belladonnæ foliorum	0 5	gr. viiss
Magnæs magnesiat q. s. ad	120 0	3iv
Misce.		

Sig.—Teaspoonful three times a day, a quarter of an hour before meals.

In cases of excessive acidity:

	Gm. or Co.	
R—Sodii bicarbonatis	60 0	3ij

Sig.—Teaspoonful in a half-glass of water, one hour after meals.

In cases of constipation with excessive acidity:

	Gm. or Co.	
R—Magnesi oxidis	20 0	3v
Sodii bicarbonatis	60 0	3ij
Misce.		

Sig.—Teaspoonful in a half-glass of water, one hour after meals.

In cases of diarrhea with excessive acidity:

	Gm. or Co.	
R—Bismuthi subcarbonatis, Cretæ præparatæ, Pulveris ossis aa	30 0	3j
Misce.		

Sig.—Teaspoonful in water one hour after meals.

In cases of subacidity the following bitter tonics:

	Gm. or Co.	
R—Tincturæ nucis vomicæ	8 0	3ij
Tincturæ cinchonæ comp. q. s. ad	90 0	3iij
Misce.		

Sig.—Teaspoonful three times a day, before meals.

	Gm. or Co.	
R—Tincturæ gentianæ compositiæ	60 0	3ij
Misce.		

Sig.—Teaspoonful in water three times a day, before meals.

In cases of deficient hydrochloric acid:

	Gm or Co	
R—Acidi hydrochlorici diluti . . .	120 0	℥iv
Misce.		
Sig.—Fill double capsule and take four such with water, at intervals of ten minutes, after each meal. (See page 281.)		

	Gm or Co.	
R—Glyceriti pepsini, N. F. . . .	240,0	℥vii
Misce		
Sig.—Tablespoonful in water during meals.		

In cases of impaired motility:

	Gm or Co.	
R—Strychninae sulphatis	0 003	gr. ʒ
Misce et ft. pil. vel tab. no. i.		
Sig.—One, three times a day, before meals.		

Surgical Treatment.—Many cases of nervous dyspepsia may require surgical intervention, and our attention must always be given to the possible presence of gallstones or chronic appendicitis. Surgeons have repeatedly called attention to the frequency with which chronic appendicitis and gallstones are associated with gastric symptoms. It is now established that appendicular disease does produce definite gastric symptoms, a condition for which Paterson suggests the term "appendicular gastralgia." (See page 691.)

Clinical experience presents strong evidence that there are gastric disturbances which are relieved or even completely dissipated by removal of the appendix. Examination of the appendices removed in association with gastralgia, pylorospasm, gastric and duodenal ulcers, cholecystitis, and cholelithiasis, shows that there is a high percentage of appendices with partially or completely obliterated lumen in all of these conditions. Macarty and McGrath found that of 365 patients on whom cholecystectomy was performed, 13 per cent. gave definite histories of pain and soreness in the region of the appendix. In 59 of these patients with cholecystitis the appendices were removed and 69 per cent. of them showed undoubted gross or microscopic evidence of inflammation, varying from a chronic catarrhal condition to complete obliteration and peri-appendicitis.

From a careful study of 271 cases of achlorhydria gastrica hemorrhagica, with a complex of gastric symptoms, Pileher found that in 156 cases the onset seemed to bear an immediate and direct relation to various diseases. In 100 of these patients operated on, the trouble in 36 was found to be due to appendicitis, in 32 to gall-bladder trouble, in 16 to gall-bladder and pancreatic disease combined, in 12 to appendicitis and gall-bladder involvement combined, and in 16 the stomach alone was found diseased. In 24 there was pylorospasm—in 18 with appendicitis and in 6 with gall-bladder

involvement. The achlorhydria is attributed to reflex inhibition of gastric secretion by disease elsewhere than in the stomach. From this it would seem that hypersecretion and hyposecretion of hydrochloric acid may be due to the same remote causes in different patients (see page 464).

UMBILICAL DYSPEPSIA.¹

A defect in the abdominal parietes preventing closure of the umbilical canal results in what is known as a congenital opening. Only when there is protrusion do we regard it as a hernia. It is a somewhat common occurrence to find the opening at the navel unobliterated. The defect is at the opening for the omphalomesenteric duct and the urachus. In the majority of instances no injury results from this non-closure. This umbilical opening may at first be very small—so small as to escape the notice of the examining physician—and later assume large dimensions. It is always congenital in origin.

All these patients have symptoms of nervous dyspepsia due to increased irritability of the autonomic nervous system. They are often free from distressing symptoms for days, and then, from some trivial cause, the symptoms recur. The appetite is apt to be capricious; coarse food may be taken without aggravation of symptoms, and then again a diet which ordinarily might be taken with impunity by a person suffering from organic disease of the gastro-intestinal tract is rejected as not agreeing with the patient.

The patient complains of fulness of the head, headache, inability to work, vertigo, lassitude, and depression. He may experience uneasy sensations one or two hours after meals, or have a feeling of heaviness immediately after eating. The degree of discomfort does not depend upon the quality or quantity of food taken. The patient is usually constipated. The nutrition is good. When the subjective symptoms become severe, loss of weight results from refusal of the patient to partake of adequate nourishment. Deep palpation with the ball of the finger over the umbilicus elicits severe pain. This may radiate in different directions, or be referred to some distant part of the abdomen. As soon as the pressure is released, the pain ceases. The stomach may function in a perfectly normal manner. In some cases, however, hyperacidity is present. The diagnosis is made by the pressure pain over the umbilicus, together with the discovery of the failure of parietal union.

The treatment consists in drawing together both sides of the recti muscles at the level of the umbilicus and securing them in this position by adhesive plaster. This induces a firmness in the

¹ Charles D. Aaron, Umbilical Dyspepsia, Transactions of the American Gastroenterological Association, 1916.

parts so that an increase in the intra-abdominal pressure will not irritate the opening at the umbilicus when the underlying organs are forced against it. The plaster may be allowed to remain two or three weeks without discomfort. After the adhesive plaster has been applied two or three times, complete relief of the digestive troubles ensues. Closure of the unobliterated opening by surgical intervention is the ideal treatment.

CHAPTER XX.

SECRETORY NEUROSES.

HYPERCHLORHYDRIA—HYPERACIDITY—SUPERACIDITY.

HYPERCHLORHYDRIA.

THE term "hyperchlorhydria" is applied to that condition of the gastric secretions in which the quantity of gastric juice is normal but the percentage of free hydrochloric acid higher than normal. The hyperacid gastric juice is secreted during digestion only, from the stimulus of food in the stomach. Some writers maintain that hyperacidity is not a clinical entity, but merely one aspect of hypersecretion. This view is in opposition to a convincing array of clinical facts and observations; we are justified in looking upon hyperacidity as a condition entirely independent of hypersecretion.

Hyperacidity is primarily a disturbance of the gastric function in which the mucous membrane of the stomach, under the stimulus of food, secretes gastric juice containing an excessive amount of free hydrochloric acid. It may be of purely nervous origin, a secretory neurosis dependent upon the abnormal stimulation or inhibition of certain nerve trunks leading to the stomach. Or it may be due to an organic disease of the gastro-intestinal tract or some remote organ which reflexly sends impulses which disturb the vegetative nervous system (see page 387). It is not always possible to draw a distinct line between the two varieties, neurotic and organic; so they may be considered together. These forms of hyperacidity are designated *genuine*, in contradistinction to those which occur secondarily as sequelæ of other pathologic processes. Hyperacidity in chronic gastritis (*gastritis acida*) or gastric ulcer may be either secondary or primary—the result or the cause. The clinical symptoms characteristic of hyperacidity are sometimes misleading; the symptoms may be present when the gastric juice is of normal acidity, as shown by examination after a test meal, or they may be absent when the test shows a marked hyperacidity. The presence or absence of subjective symptoms is doubtless due to differences in the sensibility of the gastric mucous membrane.

Etiology.—Hyperchlorhydria is of very frequent occurrence. In almost 50 per cent. of all patients suffering from digestive disorders, acidity of the gastric juice is somewhat increased. It is a disease of both sexes. While it is met with chiefly in adults, neither

the young nor the old are exempt. Persons of a nervous temperament, those suffering from neurasthenia, hypochondria, or melancholia, are apt to be its victims. Hyperchlorhydria has followed grief, worry, and mental overwork. In the majority of cases the cause is psychic. Bad habits of eating, the quick-lunch counter, insufficient mastication of food, beverages either too hot or too cold, alcohol, tobacco, highly spiced dishes, all predispose to hyperchlorhydria. It frequently accompanies gastric and duodenal ulcer and constipation. In incipient phthisis, cardiac diseases, appendicitis, uterine diseases, chlorosis, cholelithiasis, and in many other conditions, hyperchlorhydria has been noted, but the bearing of these diseases upon the excessive acidity is by no means clear.

Pathology.—No characteristic pathologic changes have been found in the few cases in which autopsy has been made.

Symptoms.—Hyperchlorhydria develops gradually. At first the patient experiences an uneasy sensation one or two hours after dinner. Later this feeling becomes aggravated into one of distress occurring from one to three hours after each meal. The subjective discomforts of the patient begin at the height of digestion, during which time the acid secretion, and especially the free hydrochloric acid, greatly exceeds the normal. The degree of discomfort at this time does not depend upon the quantity of acid so much as upon the sensitiveness of the gastric mucosa. Low degrees of hyperacidity sometimes provoke painful symptoms. The pain may last for an hour or two, or longer, and then disappear. Patients are frequently able to predict the exact time the pain or distress is likely to occur. The pains vary not only in duration but in severity, from mild distress to violent cramping seizures (pylorospasm) caused by obstruction to the outflow of the acid contents, together with violent peristaltic movements of the stomach. Patients are, as a rule, able to ease their pains by partaking of some article of food. During the painful attacks the region of the stomach is distended and sensitive to pressure. Besides the gastric pain, there are very often severe headache and vertigo. Constipation is common. The victims of hyperchlorhydria do not usually produce the impression on the observer of being very sick. They appear to be well nourished, except in cases where faulty and insufficient diet has been maintained for a long time.

Diagnosis.—The diagnosis is confirmed only by examination of the stomach contents. What remnants of food are found appear finely divided and well digested. The tests for free hydrochloric acid are positive. Clinicians calculate the normal total acidity after a test breakfast to be 40 to 60; in hyperacidity the total acidity is 75 to 80. A total acidity of 160 has been recorded. It is important to ascertain the quantity of free hydrochloric acid in every case. A disk of coagulated egg protein placed in the filtrate

of the gastric contents will become digested in a short time. Gastric contents obtained three or four hours after the test meal show that meat has been entirely digested, while starches are but slightly changed. The filtrate of gastric contents after either a test dinner or a test breakfast shows the presence of starch or large quantities of erythro-dextrin. The addition of a few drops of Lugol's solution to the filtrate will produce either a blue color or an intense dark red. The presence of the unaltered or slightly altered starches is due to the fact that hydrochloric acid begins to be secreted directly after the ingestion of food, and amylolysis is thus interrupted. The test breakfast and the test-diet stool are characteristic (see pages 94 and 131).

Prognosis.—Hyperchlorhydria or hyperacidity may yield to appropriate treatment. The prognosis is, as a rule, good, except in some very protracted and severe cases. Should there be pylorospasm, atony and dilatation of the stomach are apt to supervene.

Treatment.—The treatment in most cases is medical, though surgery is sometimes necessary. Should protracted medical treatment fail to heal a gastric or duodenal ulcer, surgery is imperative. When there is chronic appendicitis, obstructive gallstone formation, intestinal adhesions, lacerated cervix, etc., prompt surgical intervention is called for; but even after operation, medical treatment of the hyperchlorhydria should be continued.

Hygienic Treatment.—In view of the fact that hyperchlorhydria is often caused by grief, worry, or mental overwork, it would appear that the first thing to do is to regulate the daily life and habits of the patient. Business men, lawyers, physicians, clergymen, those whose labor entails great responsibility, should be sent away from their work to an entirely different environment where they may find at least temporary relief from the strain. Women in social circles must be persuaded to lead a quieter life. Patients among the wealthy leisure class who have too much time to think over their bodily functions must be given some occupation which will engage the mind. Persons with a predisposition to hyperchlorhydria should, as a prophylactic measure, avoid errors in diet, mental overexertion, and anger.

Dietetic Treatment.—The dietetic treatment is of the greatest importance in cases of uncomplicated hyperchlorhydria. In the first place, extremes of temperature should be avoided in both food and drink. Food should be eaten slowly and thoroughly masticated, not only to facilitate salivary digestion, but to avoid irritating the stomach mechanically. All substances that are likely to irritate the gastric mucosa must be eliminated from the dietary. All kinds of acids, including the organic, such as citric, tartaric, and acetic, must be forbidden; also spices of all kinds—pepper, mustard, horseradish, etc. The salt-free diet should be instituted in every case of persistent hyperchlorhydria, to eliminate

the chlorin. Whisky and wines are in the prohibited list. To season the food the following chlorin-free inorganic salt mixture can be used as a substitute for common salt:

	Gm.
Dicalcium phosphate	5 8
Monomagnesium phosphate	3 4
Dipotassium phosphate	7 7
Potassium citrate	1 7
Sodium citrate	7 4
Calcium lactate	4 0
Mix and pulverize.	

Jacobson recommends fresh meat, potatoes, oatmeal, carrots and cauliflower cut fine, to be boiled for hours with several changes of water; stewed apples, prunes and apricots; very weak tea and coffee; butter freed from salt by washing small particles thoroughly in running water; one egg and about 50 Cc. of milk or cream per day, but no more. Distilled water is used for drinking. A sample diet for one day might be as follows:

Breakfast.—Oatmeal gruel with sugar and a little cream; apple sauce; very weak coffee with sugar and cream.

Dinner.—Fresh meat, boiled and hashed; potatoes boiled and mashed; carrots likewise; special salt-free butter; orange juice diluted and sweetened.

Supper. One egg, raw, boiled or poached; boiled rice; purée of prunes; very weak tea with sugar and cream.

The food should be rich in protein and as poor as possible in starchy substances. The total acidity of the gastric secretions is much greater with a protein than with a carbohydrate diet, but the amount of free hydrochloric acid is much less. Owing to the large percentage of extractives in meat which excite the flow of gastric juice, it seems advisable to substitute some other form of protein, as eggs, milk, cheese, or vegetable protein. However, when meat is prescribed, it should be well cooked to remove the extractives. Raw meat should be avoided, owing to its excessively stimulating effect on gastric secretion. Oatmeal, aleuronat meal, bread and cocoa, all of which are rich in protein, are useful food substances in the treatment of hyperchlorhydria.

Carbohydrates should not be eliminated entirely from the diet, but should be restricted. They may be taken in finely divided form; that is, vegetables such as spinach and cauliflower must be taken as purée. Salads and fresh fruits are to be avoided. When free hydrochloric acid appears early, interrupting the digestion of carbohydrates, amylolysis may be assisted by the use of dextrinated carbohydrates (zwieback, toast). Sugar has been found valuable in the dietary of hyperacidity, inasmuch as concentrated saccharated solutions diminish not only the total acidity, but likewise the free hydrochloric acid, to a marked degree. Sugar may be given in a variety of forms, such as sweet dishes, jellies, jam, and honey.

Fats fulfil the same rôle as sugar. It has been demonstrated that fat not only hinders gastric secretion, but diminishes the quantity of free hydrochloric acid. Fats can be given after it has been ascertained that they do not disturb the motility of the stomach or interfere with the assimilation of other foods. Bacon provides fat in an agreeable form. Milk, cream and butter are indicated.

Oils of various kinds have been employed with good results in the treatment of hyperchlorhydria. Olive oil, given in connection with the usual test breakfast, decreases the gastric acidity at the end of the hour and retards the evacuation of the stomach. The action of oil on the gastric functions is only a temporary one. It has no effect on subsequent meals unaccompanied by oil. The therapeutic value of oil is apparent. In suitable cases it is preferable to antacids because of its caloric value. In hyperchlorhydria it should precede the meal.

Wiley says that cottonseed oil may be safely substituted for olive oil. When used with salt on bread it makes a very acceptable substitute for cream and butter, and is free from the germs of the diseases we contract from the animal world. Not only is cottonseed oil more easily digested than corn oil, peanut butter, or even olive oil; it does not ferment in the stomach.

Large quantities of fat are particularly indicated in cases of hyperacidity accompanied by constipation. On the other hand, a purely lactovegetable (see page 422) or meat-poor regimen is recommended in pronounced nervous forms of hyperacidity; the vegetables should be thoroughly cooked and finely divided.

Regarding the frequency of meals, it is advisable to eat five or six times a day, three heavy and two or three light meals.

OUTLINE OF DIET IN HYPERCHLORHYDRIA (EINHORN).

		Calories.
7.30 A.M.	Two eggs, 50 Gm.	160
	White bread, 50 Gm.	128
	Butter, 20 Gm.	163
	Milk, 250 Cc.	169
10.30 A.M.	Matzoon or milk, 200 Cc.	135
	Crackers or bread, 30 Gm.	77
	Butter, 10 Gm.	81
1.00 P.M.	Broiled meat, 100 Gm.	210
	Mashed potatoes, 50 Gm.	63
	Bread, 30 Gm.	77
	Butter, 10 Gm.	81
	Weak tea or Vichy water, 200 Cc.	
3.30 P.M.	Same as at 10.30 A.M.	293
6.30 P.M.	Soup (with barley or vermicelli), 200 Gm.	100
	Bread and butter (bread, 30 Gm.; butter, 10 Gm.)	158
	Meat broiled or cooked, 100 Gm.	210
	Potatoes, baked, 50 Gm.	60
	Green vegetables (spinach, green peas), 50 Gm.	80
	Coffee (half milk), 100 Cc.	34
10.00 P.M.	Oysters and crackers, or cold meat sandwich	200
		<hr/> 2539

Beverages taken at meals are harmless, inasmuch as they dilute the gastric juice. Alkaline mineral waters, Vichy, and California Seltzer waters containing no carbon dioxide, may be prescribed in large quantities. Beer and coffee should be avoided. Cocoa and weak tea well diluted with milk are permissible. Pure milk, however, is the ideal beverage in these cases.

Medicinal Treatment.—Astringents.—The astringents are among the most valuable drugs we have in the treatment of hyperchlorhydria. Their action is confined to the gastric mucosa. They diminish the intensity of local inflammation. Astringents are therefore indicated in the treatment of chronic gastritis when the inflammatory process is superficial. Owing to their inhibitory effect upon secretion they are contra-indicated when the acid secretion is normal, subnormal, or absent.

Among the most valuable astringents are the salts of bismuth and silver. The physical effect of bismuth subnitrate is the formation of a protective layer over the gastric mucosa, which is particularly desirable where abrasions exist. Bismuth inhibits gastric secretion; the subnitrate materially diminishes the quantity of free hydrochloric acid.

The inhibitory effect of bismuth subnitrate is due to the nitric acid formed by the action of hydrochloric acid upon it. Bismuth subnitrate is indicated in the treatment of hyperacidity and hypersecretion (see page 265).

Recent experiments show that the silver compounds (silver nitrate, protargol, alhargin) diminish the quantity of gastric juice in inflammatory conditions of the gastric mucous membrane, but slightly increase the amount of hydrochloric acid. In the absence of inflammation, however, the silver salts were found to increase gastric secretion. Nitrate of silver, accordingly, would be indicated in cases of gastritis with either normal or subnormal acidity, rather than in hyperacidity or hypersecretion. I have obtained good results, however, from the use of silver nitrate in hyperacid conditions, whether accompanied by catarrh or not, and also in hypersecretion. Lavage with 1:1000 nitrate of silver will promptly relieve the severe pains and often abate the annoying symptoms. Nitrate of silver should be given in solution (1:750 to 1:1000), one tablespoonful of the solution in a half-glass of distilled water before meals (see page 267).

Atropin.—The effect of astringents is directly upon the gastric glands. Atropin acts through the autonomic nervous system, inhibiting gastric secretion, and diminishing the quantity of hydrochloric acid in the gastric juice without interfering with the secretion of pepsin. Atropin, furthermore, acts as an antispasmodic and analgesic; it diminishes the sensibility of the sensory nerves. Atropin sulphate in doses of $\frac{1}{2}$ to 1 milligram ($\frac{1}{16}$ to $\frac{1}{4}$ grain), given in the form of tablets, is a useful agent for promptly relieving

the painful attacks of pylorospasm. When atropin is to be administered over a long period of time it is best given as extract of belladonna, 0.02 to 0.03 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain) three times a day, before meals; or it may be advantageously given with astringents and alkalis. Atropin is a poison, and when it is necessary to secure its therapeutic effect for a considerable length of time some relatively harmless substitute should be considered. Among the less poisonous substitutes we have eumydrin, which is supposed to be fifty times less toxic than atropin (see page 271). In doses of 1 to 3 milligrams ($\frac{1}{60}$ to $\frac{1}{20}$ grain) eumydrin can be given for some little time without producing any severe general disturbances. It is very satisfactory in gastric neuroses with hyperchlorhydria and increasing gastric pains.

	Gm. or Gm.	gr.
R—Eumydrin	0.04	$\frac{1}{3}$
Sacchari	6.0	3iss
Misce et ft. pulv. no. xx.		
Sig. One, three times a day, before meals		

Alkaloids.—Of the alkaloids, codein is the only one besides atropin that does not occasion untoward after-effects. Morphin, after temporarily inhibiting secretion, is apt to cause a very copious flow of hyperacid gastric juice. Dionin and pilocarpin immediately increase the secretion. Codein may be given in doses of 0.01 to 0.03 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain) with extract of belladonna or with alkalis and astringents (see page 271).

Hydrogen Peroxid. It has recently been shown that hydrogen peroxid, taken internally, reduces the total acidity and especially the proportion of hydrochloric acid. A teaspoonful of hydrogen peroxid may be taken in a glass of water after meals (see page 272).

Magnesium peroxid has been found useful for diminishing hyperacidity. The dose is 1 Gm. (15 grains) three times a day (see page 273).

Analgesics. The following analgesic agents have been found efficacious: Cannabis indica extract, 0.01 to 0.05 Gm. ($\frac{1}{4}$ to 1 grain) three times daily; chloral hydrate; and chloroform water (1:200). Cocain is efficacious in painful vomiting. Menthol and validol act like cocain. The bromids are occasionally very useful in the nervous form of hyperacidity; bromid of strontium, 2 to 4 Gm. (30 to 60 grains) daily, is often valuable.

Acids. Hyperacidity can frequently be relieved by giving large doses of hydrochloric acid, which exerts an inhibitory influence upon the motor reflexes of the stomach. Hypermotility may induce hyperacidity, and medicinal hydrochloric acid arrests the rapid emptying of the stomach into the duodenum. The acid should be administered before meals. These patients tolerate the usual doses of hydrochloric acid very well, in contradistinction to those suffering from gastric ulcer; this fact is of value in differential diagnosis.

Alkalis. Alkalis are the remedies that are employed most frequently in the treatment of hyperchlorhydria. Experimental research concerning the effect of alkalis in the stomach has established the fact that, reaching the stomach in sufficient quantity, they are capable of neutralizing the hydrochloric acid secreted. Magnesium oxid and sodium bicarbonate serve this purpose. Sodium bicarbonate not only neutralizes the acid, but diminishes the secretion; but magnesium oxid is capable of neutralizing a greater amount of acid—in the same dose four times as much. In hyperacidity and hypersecretion these drugs should be given in large and repeated doses (see Chapter XIII): sodium bicarbonate, 4 to 8 Gm. (5j-ij); magnesium oxid, 1 to 2 Gm. (15 to 30 grains). A soluble aluminum silicate, capable of slowly combining with hydrochloric acid, is sold under the trade name neutralon. It is a white, odorless and tasteless powder, insoluble in water, and has been found useful in hyperchlorhydria, hypersecretion, and gastric ulcer. The dose is one teaspoonful 15 to 30 minutes before meals.

In cases of gastric ulcer with symptoms of hyperacidity it is advisable to avoid the carbon dioxid alkalis, on account of the gastric distention that is likely to follow the formation of gas.

The Carlsbad salts fulfil indications similar to those for the other alkalis mentioned. Natural crystalline Carlsbad sprudel salt consists (after removal of the water of crystallization) of sodium sulphate, 98.79; sodium carbonate, 0.45; and sodium chlorid, 0.76. The artificial salts (see page 264) are similar in their effects; but, while they are cheaper, they are not so agreeable to the taste as the natural salts.

Magnesia magma (milk of magnesia) is a suspension of magnesium hydroxid in water. A dose of one-half to two tablespoonfuls will neutralize the acid in hyperacidity, and will act favorably when this condition is complicated with constipation.

In cases of hyperacidity the proper times for the administration of alkalis are directly after eating, and at the height of digestion when the secretion of acid is freest. Patients are frequently able to tell this particular moment with considerable exactness, as it coincides with the onset of their painful symptoms.

In the absence of saliva, jaborandi or pilocarpin can be given, since these drugs are known to be sialagogues. The absence of salivary secretion retards amylolysis greatly, and proteolysis as well. In cases of such pronounced hyperacidity that salivary digestion is inhibited, malt diastase combined with alkalis acts well. I think, however, that in such conditions the object could be better accomplished by more prolonged mastication and insalivation of the food.

Course of Medication. The course of medication in hyperchlorhydria is as follows: In light cases the attempt is made with alkalis

alone. When the cases are more obstinate and cause much discomfort, astringents may be given in addition to the alkalis. Severe cases, especially those with excruciating pains, a high degree of acidity, and pylorospasm, require the administration of the alkaloids, combined with alkalis and astringents.

Lavage of the Stomach.—In cases of hyperchlorhydria complicated with atony or disturbances in motility, lavage of the stomach is useful. Lavage may be performed late at night after an early supper, or early in the morning. It should be followed by a solution of Carlsbad salt or a 1:1000 solution of nitrate of silver, to be washed out with pure water.

Physiotherapeutic measures are indicated as palliatives in severe cases only, where something must be done at once. Hot compresses over the region of the stomach mitigate the severity of pain. The Leiter coiled tube (Fig. 61) has a quieting and anti-spasmodic action.

In chlorotic patients, when iron is to be given, I prefer the hypodermic administration of the citrate of iron, as described on page 581.

The following formulas for combinations of alkaline medications will be found serviceable in various conditions:

	Gm or ℥.	
R Sodii bicarbonatis,		
Magnesi oxidi	ss 4 0	℥j
Calci carbonatis	6 0	℥iiss
Misce et ft. pulv.		

Sig. Take one teaspoonful immediately after each meal, with water. The dose may be repeated, increased or diminished as required.

	Gm or ℥.	
R—Magnesi oxidi	10 0	℥iiss
Sodii bicarbonatis	40 0	℥x
Misce et ft. pulv.		

Sig. One-half to one teaspoonful three times a day, one or two hours after meals, in half a glass of water.

	Gm or ℥.	
R Bismuthi subnitratu,		
Magnesi oxidi,		
Sodii bicarbonatis	ss 30 0	℥j
Misce et ft. pulv.		

Sig. —Heaping teaspoonful in a half-glass of water one hour after meals.

	Gm or ℥.	
R—Magnesi oxidi,		
Pulveris radici rhei	ss 20 0	℥v
Sodii bicarbonatis	40 0	℥x
Misce		

Sig —One-half to one teaspoonful in water one or two hours after meals.

For hyperacidity with diarrhea:

	Gm or ℥.	
R—Crete preparata,		
Bismuthi subgallatis,		
Sodii bicarbonatis	ss 10 0	℥iiss
Misce et ft. chart. no. xx.		
Sig —One powder six or more times daily.		

For hyperacidity with pain:

	Gm. or Cc.	
R—Codeinæ phosphatis	0 25	gr. iv
Crete præparatæ	4 0	℥i
Bismuthi subnitratæ	10 0	℥iiss
Magnesiæ oxidæ	4 0	℥i
Sodii bicarbonatis	10 0	℥iiss

Misc et ft. chart. no. xv.

Sig.—One powder to be taken one hour after meals.

	Gm. or Cc.	
R—Gocainæ hydrochloridi	0 10	gr. iss
Heroinæ hydrochloridi	0 02	gr. $\frac{1}{2}$
Atropinæ sulphatis	0 01	gr. $\frac{1}{4}$
Extracti ergotæ	1 0	gr. xv
Aquæ destillatæ	10 0	℥iiss

Misce.

Sig.—Five to twenty drops every hour until relieved.

If between meals there is burning or pain in the stomach due to hyperchlorhydria, Stockton's gastric sedative formulæ are useful (see page 414).

CHAPTER XXI.

SECRETORY NEUROSES (CONTINUED).

HYPERSECRETION — GASTRORRHEA — GASTROSUCCORRHEA — GASTROCHYLORRHEA.

The term "gastrosuccorhea" was introduced into medical literature as representing a clinical entity in 1882, by Reichmann. In gastrosuccorhea, gastrorrhea, or hypersecretion, the glands of the stomach secrete gastric juice constantly; considerable amounts may be found in the fasting stomach, before the first meal of the day. Opinions vary as to the quantity of gastric juice that indicates hypersecretion. The percentage of hydrochloric acid may or may not be above the normal.

INTERMITTENT OR PERIODIC HYPERSECRETION (ACUTE INTERMITTENT GASTRORRHEA).

Etiology.—In ascertaining the cause of hypersecretion we must look to the nervous system. Among the etiologic factors may be a derangement in the vegetative nervous system, manifested in neurasthenia, hysteria, anger, worry, and mental overexertion. Young adults are particularly prone to attacks. It is highly probable that chronic hyperacidity may induce acute hypersecretion, especially when the gastric mucous membrane is being irritated. A perfectly healthy mucous membrane with habitually normal secretion may, however, produce a flow of hyperacid gastric juice on the ingestion of certain articles of diet. Very cold beverages may occasion hypersecretion. Acute hypersecretion occurs not infrequently after the healing of gastric ulcer; the exciting cause is presumed to be the cicatrix of the ulcer.

Symptoms.—Hypersecretion appearing at regular intervals is characterized by violent pain and copious vomiting of acid materials. As a rule the seizures are sudden and not anticipated by the patient; they occur mostly during the night or in the early morning hours. After the expulsion of food remnants, the vomited matter consists of varying quantities of greenish to clear watery fluid which exhibits all the characteristics of gastric juice. The chemical tests show the presence of hydrochloric acid and pepsin. The microscope indicates the presence of epithelial cells and leukocytes. The violent pains, together with the retching and vomiting, reduce the patient to a condition of exhaustion. There is pronounced pallor, perspiration is free, and the pulse is feeble and rapid; appetite fails, and the bowels are torpid; the urine is scanty and usually alkaline in reaction. The attacks may be of great severity,

or they may be very slight; their duration varies from one or two hours to as many days. Convalescence is usually rapid, and the patient may feel well enough to resume his occupation the day following the attack.

Not infrequently the seizures are accompanied by violent headaches. Attacks in which headache is a prominent symptom have been designated by the special term *gastrocynsis*; they belong, however, to the class of intermittent hypersecretion. Sometimes the cephalalgia is of such a character that the local gastric disturbance is obscured. The patient may feel perfectly well during the interval between the seizures; slight gastric discomforts, such as pressure, fullness, eructations, are, however, experienced after eating. An examination of the stomach contents during the interval between attacks shows an excessive acidity, which would indicate the possibility of a coexistent chronic hyperacidity.

Diagnosis.—The diagnosis is confirmed by emesis of large quantities of liquid which responds to the tests for gastric juice. Analysis of the gastric contents yields characteristic results (see page 94).

Treatment. When the physician is called during an acute attack of hypersecretion, it is his first duty to cut short the attack, or, failing in this, to mitigate its severity. At its onset the disease may be diminished in severity, or even aborted, by the administration of large doses of sodium bicarbonate or magnesium oxid. Stomach lavage is indicated either with clear water or with water containing nitrate of silver (1:10,000). The drinking of milk has sometimes a salutary effect. Should the attack continue, atropin, 1 milligram ($\frac{1}{60}$ grain), is indicated, to be administered hypodermically. This is the most reliable medicament. Morphin and atropin may be combined and administered subcutaneously. Suppositories of extract of belladonna combined with morphin are useful, but do not act so promptly as atropin and morphin hypodermically. The abdominal pains are to be treated with hot compresses, moist or dry. No food should be taken. Thirst should be allayed by small pieces of ice in the mouth. In the absence of distressing symptoms during the intervals between the attacks, and especially if the secretion of hydrochloric acid is normal, a bland diet may be prescribed. Irritating food, the use of tobacco, and excessive mental efforts, should all be avoided. In some cases all therapeutic measures fail to prevent a recurrence of the attacks and they become chronic. In the treatment of nervous patients suffering from chronic hypersecretion, favorable results are frequently obtained by a sojourn in a high altitude.

CONTINUOUS HYPERSECRETION (CHRONIC GASTRORRHEA).

In this form of hypersecretion the stomach secretes, apparently spontaneously, at any rate without the stimulating influence of

food, a strong digestive juice, and that continuously. Normally only a few milliliters of fluid contents are found in the fasting stomach, and pepsin and hydrochloric acid are either absent altogether or present in small quantities. In cases in which large quantities of gastric juice are found regularly on removing the contents of the stomach after prolonged abstinence from food, the diagnosis of chronic gastrorrhea is confirmed.

Etiology. Chronic hypersecretion may develop from a previously existing hyperacidity, which explains the frequent simultaneous occurrence of both disease processes. In such instances hypersecretion is an aggravated form of hyperacidity, in which the secretory and sensitive condition of the mucous membrane is more pronounced than in cases of uncomplicated hyperacidity. There can be no doubt that nervous influences, too, constitute an important factor in the causation of hypersecretion. The majority of cases of chronic hypersecretion occur in youth and middle age and in males. The secretion of gastric juice is augmented by the abuse of alcohol and tobacco. Among the causative factors are to be enumerated dietetic errors and mental perturbations. Gastric ulcer is also a cause of chronic hypersecretion. The frequent coincidence of chronic gastrorrhea and atony of the stomach is worthy of note. There is also the possibility of a relationship between hypersecretion and the traction of hernias on the linea alba and traction on the mesenteries in gastroenteroptosis. Possible derangement of the vegetative nervous system must not be overlooked (see page 387).

Symptoms.—Chronic hypersecretion is characterized by slow onset, with mild symptoms, pressure and fulness after eating, eructations, and pyrosis. The prodromes are those of chronic gastritis. The symptoms may disappear, only to recur in aggravated form. Pain is an additional symptom; according to the statements of the patient, it does not follow the ingestion of food. Pains may, however, be induced by partaking of food, or occur suddenly at irregular intervals. Thus the pain of hypersecretion differs from that of hyperacidity, which usually comes on at the height of digestion. The fact that, the stomach being empty, the ingestion of food while the pain is most severe will bring relief, is of diagnostic importance. At the height of a paroxysm, vomiting frequently occurs, and it has a marked effect in mitigating the severity of the pains. The greenish watery fluid expelled from the stomach may amount to several liters. Hematemesis is sometimes noted; when it is present, gastric ulcer or erosions of the stomach should be borne in mind. The appetite is usually fair, but suffers as the pains become more persistent and severe. The quantity of food taken by the patient becomes less and less, with the result that he loses weight and flesh. In pronounced cases of chronic hypersecretion the patient complains of thirst, the bowels

are constipated, and the urine is turbid from a slight degree of alkalinity.

Many cases of hypersecretion are complicated with atony and motor insufficiency of the stomach. Such cases are characterized by the vomiting of large quantities of fluid.

Diagnosis.—On the removal of the stomach contents in a well-marked case of hypersecretion six or seven hours after a test meal, large quantities are found, with no meat remnants, but residues of starchy materials, which are precipitated to the bottom of the vessel. The total acidity of the material removed is often high, 90 to 100, and free hydrochloric acid is increased from 50 to 70. Sometimes, especially in cases of hypersecretion accompanied by dilatation of the stomach, the contents show copious evolution of gas in the fermentation tubes kept in the incubator (Fig. 15). If the stomach be carefully cleansed at night and the patient instructed to fast, removal of the stomach contents in the morning will show varying quantities of liquid secretion (up to $\frac{1}{2}$ liter) possessing the properties of gastric juice. A positive finding of this kind serves to confirm the diagnosis of hypersecretion (see page 94).

External Examination of the Stomach.—Palpation reveals an accelerated peristaltic motion. A thickened pylorus may be sometimes felt by the palpating hand, inasmuch as the pyloric exit of the stomach is often in a state of tetanic contraction induced by the large quantity of acid present. As soon as a portion of the acid stomach contents passes through into the small intestine the pylorus closes, so that it is impossible for the stomach to properly empty itself. Each relaxation of the pylorus is followed by a spasm which blocks the exit (pylorospasm). The muscles of the stomach, meanwhile, attempt to force a passage by means of increased peristaltic movements. This vicious circle is the cause of the gastric dilatation which so frequently complicates these cases. Thickening of the pylorus may be the result of an old cicatrix from healing of a gastric ulcer (see page 479). A Roentgen-ray examination assists in the diagnosis.

Prognosis.—The prognosis for complete recovery from chronic hypersecretion is not always favorable. Recovery may be anticipated only in that class of patients who are in a position to continue treatment for a long period of time. Patients who are unable to take the necessary care of themselves are apt to have relapses after intervals of improvement.

Treatment.—The treatment of chronic hypersecretion is clearly indicated on examination of the contents of the stomach, and by the subjective and objective symptoms. Should there be evidence of any other pathologic condition complicating or maintaining the hypersecretion, this must receive due consideration. If gastric ulcer is present it must receive attention; and the neurasthenic requires special treatment. Patients suffering from hyperacidity

should be so treated as to preclude the possibility of transforming that condition into hypersecretion. Anger, excitement, mental shock, and improper diet should be avoided as much as possible. Frequently, however, hypersecretion would seem to be an idiopathic disease, one for which there is no assignable cause.

Diet.—The chief factor in the therapeutics of hypersecretion is a properly selected dietary. Since the quantity of gastric juice secreted during the period of digestion is abnormally large, proteolysis is likely to be satisfactory. This is attested by the fact that when the stomach contents are removed after a meal of meats and starches, scarcely any meat remnants remain; the residue is made up principally of amylaceous material. The gastric digestion of carbohydrates is held completely in abeyance in hypersecretion, since the ptyalin is neutralized almost as soon as the food reaches the stomach. From this it follows that the food should be mainly protein. So far as the quantity of fats in the food is concerned, the statements regarding fat in hyperacidity hold good. Fat diminishes the secretion of hydrochloric acid, and should therefore be employed extensively in the treatment of hypersecretion. A diet of protein and fat is indicated. The gastric mucous membrane is in a condition of chronic irritation; therefore, in prescribing diet, care must be exercised to avoid articles of food which are apt to aggravate this condition. All spices, acids, and highly seasoned foods must be eliminated from the diet. Extremes of temperature in foods and beverages should be avoided. Thorough mastication of the food is an important requirement; the food should be in a finely subdivided condition before being swallowed.

The various kinds of meat may be taken by this class of patients. Meats should be well cooked, since the extractives in rare meat excite the secretion of still greater quantities of gastric juice. Soft-boiled eggs, scrambled eggs, omelet, and cream cheese are indicated. Of fats, numerous articles merit consideration; for example, butter, olive oil, sesame oil, cottonseed oil, milk and cream, cocoa, and yolk of egg. Milk is an excellent liquid food; it is non-irritant and has a neutralizing effect upon the acidity of the gastric juice.

Carbohydrates, for obvious reasons, should be restricted unless they have been dextrinized; wheat bread should be eaten in the form of toast. Crackers and zwieback are suitable articles of diet. Carbohydrates should be given in the form of leguminous flour soups, or gruels, or as sago and oatmeal. The patient may partake of a small quantity of mashed potatoes. All green vegetables should be prohibited. Sugar is allowable only in cases in which the motility of the stomach is normal, since it may give rise to excessive fermentation. Care should be exercised in the preparation of dishes for this class of patients, to avoid even a moderate use of condiments.

Number of Meals.—Regarding the number of meals, a good rule to follow is: Eat often, and a little at a time. The object is to take up the gastric juice as fast as it is secreted. Frequent administration of food will tend to bring about an entire cessation of pain; suitable articles of food should be at hand all the time. Milk, biscuits, and hard-boiled eggs should be easily accessible to the patient on retiring at night; these taken at the beginning of a pain will often suppress it.

Liquids.—Liquids should be taken in moderation, since they tend to increase the quantity of fluid in the stomach. They are particularly harmful in cases of hypersecretion combined with atony or dilatation. Alcohol and coffee should be avoided. When there is great thirst, and it is inadvisable to partake of sufficient liquid by mouth to allay the thirst, a small enema (150 Cc.) of physiologic salt solution will satisfy the craving.

In severe cases of hypersecretion it is sometimes advisable to resort to rectal feeding for a period of eight to ten days (see page 243). By this means irritation of the stomach by food will be obviated, and a diminished secretion of gastric juice will result. An exclusive milk diet for eight to ten days is very beneficial; this is what is called the "milk cure." The milk diet acts as a sedative to the sensory nerve endings in the gastric mucous membrane. Inter-current diarrheas may be prevented by the addition of lime-water to the milk in the proportion of 1 to 3 or 1 to 4. The feedings should consist of 350 to 400 Cc. (12 to 14 ounces) of milk every two hours, or a daily amount of 2800 Cc. (about 3 quarts). The required number of calories may be attained by the addition of a milk-cream mixture.

In cases of hypersecretion with pylorospasm, the continuous saline instillation—the Murphy drip (see page 239)—to restore the needed water to the tissues, is beneficial. This treatment has a distinctly favorable influence on the spasm, promoting relaxation, as evidenced by the cessation of vomiting. Systematic continuous proctolysis, as a direct means of influencing the pylorospasm in addition to its other advantages, is recommended.

Medicinal Treatment.—The alkalis are valuable therapeutic agents in the treatment of hypersecretion, as well as in hyperacidity. They may be prescribed to be taken before, during, or after the ingestion of food. Given during or before a meal, they are calculated to facilitate amylolysis, since they neutralize the free hydrochloric acid which would otherwise put a stop to the action of the ptyalin of oral digestion as soon as it reached the stomach.

Alkalis are given after meals to neutralize excessive acidity that is producing painful symptoms. Large doses have the effect of immediately relieving the pain. They are valuable for allaying the violent paroxysmal nocturnal pains of hypersecretion, but unfortunately the relief is not permanent. Magnesium oxid and

sodium bicarbonate are particularly useful in these conditions. The administration of the Carlsbad salt in the morning after the night's fast is a useful procedure, since it neutralizes the gastric secretion and washes it into the duodenum (see page 264).

For the improvement of amylolysis, artificial salivary ferments have been employed. One of these is an artificial ptyalin. This preparation may be given with the alkalis. Again, we have taka-diastase and malt diastase; these act in the same way as ptyalin. Taka-diastase may be prescribed to be taken with the food, with or without alkalis, in cases of hyperacidity and hypersecretion. Panase is a pancreatic preparation similar in its action.

Atropin sulphate, 1 milligram ($\frac{1}{60}$ grain), has been given hypodermically during violent attacks of pylorospasm. The object is to restrict the secretion. In many cases the following treatment, kept up for fourteen days, will be sufficient to effect a permanent cure:

	Gm. or Co.
R—Atropinæ sulphatis	0.0005 0.001 gr. $\frac{1}{60}$ - $\frac{1}{30}$
Misce.	

Sig.—For subcutaneous injection three times a day, and always by the physician himself.

The contra-indications are affections of the heart and blood-vessels. If accommodation paresis, dry sensation in the throat, and vertigo are not very severe, the treatment may be continued. A test for the applicability of the atropin treatment consists in giving on two succeeding days 0.0005 or 0.001 Gm. ($\frac{1}{60}$ to $\frac{1}{30}$ grain) of atropin subcutaneously shortly before the test breakfast. If, on examination, the secretion is found to have diminished, the treatment is indicated. Eumydrin also can be used, and is safer. Favorable results have been secured from the use of extract of belladonna in suppository form. This drug may also be given internally, either alone or in combination with the alkalis (see page 271).

Astringent remedies are to be employed as in the treatment of hyperacidity. Preparations of bismuth or nitrate of silver may be given by mouth (see pages 265 and 267).

Lavage of the Stomach.—When the symptoms do not yield to diet or drugs, the stomach should be washed out just before the evening meal; it is then in a condition to receive and to digest a small supper. The acid secreted after the supper is neutralized by the food. Lavage is also indicated in the morning before breakfast, to remove the acid secreted during the early morning hours, as well as the remnants of food that may have remained overnight in the stomach. In pronounced cases of hypersecretion, lavage at these two periods is imperative; painful attacks are often cut short by a single washing. Pure water, lukewarm, is employed in the process, to be followed by lavage with a mild alkali, such as sodium bicarbonate solution. Lavage with 1:10,000 nitrate of silver or with a suspension of bismuth subnitrate has been employed with good success.

Mineral Waters.—Carlsbad water, taken in large doses and for a long period of time, has the effect of diminishing the secretion of gastric juice. It is favorable to the peristaltic movements of the stomach, and tends to diminish the sensitiveness of that organ. The waters of Saratoga are similar in action to the Carlsbad waters (see Chapter XII). Vichy water may be prescribed for neurotic patients.

Physical Treatment.—Massage, vibration, and electric treatment must not be employed in hypersecretion. Hydrotherapeutic and thermic applications may, however, be made extensively and to good advantage. Hot compresses, moist or dry, are particularly adapted to the treatment of painful seizures. In severe cases prolonged rest in bed is essential.

Surgical Treatment.—In cases of hypersecretion complicated with ulcer of the pylorus or duodenum, marked motor disturbances and stenosis of the pylorus, gastroenterostomy may prove of permanent benefit.

ALIMENTARY HYPERSECRETION.

Alimentary hypersecretion is a less severe variety of chronic gastrorrhea. While in chronic gastrorrhea the gastric mucous membrane is in a state of continuous irritability, as a result of which the gastric secretion is constant and abnormal in quantity, even when the stomach is empty, in alimentary hypersecretion the symptoms, which afford the same clinical picture when they appear, are induced only by stimulation of the gastric mucous membrane. There must be a stimulus, however slight, before the abnormal secretion begins. Much less stimulation is required, however, than in the normal stomach; the secretion begins sooner.

Symptoms.—The subjective symptoms are less severe than those of chronic gastrorrhea, resembling more closely those of hyperacidity. Gastric discomforts, consisting of pyrosis, pressure, acid eructations, and pain of greater or less severity, follow almost immediately the ingestion of food; whereas in hyperacidity these symptoms do not appear until some little time after eating. In contrast with hyperacidity, the discomforts of alimentary hypersecretion are not diminished by partaking of food. The appetite is usually good, though patients often become poorly nourished because they are afraid to eat. When alimentary hypersecretion is complicated with motor disturbances, the distressing symptoms persist while the food remains in the stomach. With intense attacks of gastric pain, as in chronic gastrorrhea, constipation is a frequent concomitant symptom.

Diagnosis.—Palpation of the empty stomach, as a rule, does not reveal anything of note. When the stomach is filled with food, palpation occasionally causes a slight degree of pain. Splashing

sounds can be elicited occasionally during the height of digestion especially if atony be present. The diagnosis must be made by means of the test meal or test breakfast. The facts that the fluid portion exceeds the solid residues, and that the total quantity of fluid removed is greater than the amount introduced, are of diagnostic importance. The quantity of free hydrochloric acid may be found above normal. There are no pathologic findings which can be said to be pathognomonic of this disease.

Treatment.—The dietetic treatment is the same as that prescribed for chronic gastrorrhea. Owing to the fact that amylolysis is deficient, the food should be chiefly of a protein and fatty nature. It should be finely subdivided, preferably mucilaginous in consistency; and the meals should be limited to three a day and taken at regular intervals, in order to prevent, to the greatest possible extent, irritation of the gastric mucous membrane. In these cases, too, it is sometimes expedient to place the patient on an exclusive milk diet, keeping him in bed.

Medicinal Treatment.—Alkalis are to be employed extensively both before and during meals, to assist amylolysis, and also after meals at the height of digestion. Atropin sulphate, hypodermically by mouth, or by suppository, is of value when there is pain. Occasionally it is necessary to continue the administration of atropin over an extended period. Astringents are also indicated.

Treatment by Lavage of the Stomach.—The best time for this procedure in cases of alimentary hypersecretion is late at night after an early supper, in order to relieve the stomach of food remnants and thus prevent gastric secretion during the night. Lavage with pure water may be succeeded by lavage with alkalis or with a solution of nitrate of silver (1:10,000).

CHAPTER XXII.

ACUTE GASTRITIS—SIMPLE, INFECTIOUS, TOXIC, PHLEGMONOUS.

ACUTE gastritis (acute gastric catarrh) is an inflammation of the gastric mucous membrane accompanied by disturbances of digestion. The inflammation may be simple, infectious, toxic or phlegmonous. It may be limited to the superficial layer of the gastric mucosa, or it may involve the glandular epithelium, the parenchyma, or the interstitial tissues.

SIMPLE ACUTE GASTRITIS.

This is the form that is most frequently encountered in general practice. No age or class is exempt.

Etiology.—Among the etiologic factors are: errors in diet—an excessive amount of food taken at one time; mechanical, thermic or chemical irritants; foods highly spiced or fermented; unripe or overripe fruits; cold drinks, soda water and ice-cream; food in process of decomposition; the excessive use of condiments; and overindulgence in alcohol.

The tendency to acute gastritis is greater in some individuals and families than in others. In many persons the predisposition is such that the slightest excess in diet precipitates the catarrhal evolution. In this class are anemic women, invalids, and elderly persons. Acute gastritis may be secondary to other affections, as the acute infectious diseases, typhoid, variola, pneumonia, or measles.

Toxic gastritis in its milder forms may be placed in the category of simple acute gastritis. Decomposition products, such as spoiled food, meat or cheese, are contributory to this form of gastric catarrh. With acute gastritis may be classed the light forms of acute infectious gastritis caused by microorganisms introduced with decomposed food. It is well known that parasites, oxyurids, teniae, ascariides, and larvae of flies, taken into the stomach, may cause gastritis.

Pathology.—The gastric mucosa is wholly or partially swollen and reddened, the inflamed portions covered with tenacious mucus. In occasional instances there are slight hemorrhages. The submucosa may be edematous. Microscopically, the surface epithelium appears altered; it is swollen, opaque and desquamated. Similar changes are noted in the glandular epithelium. The capillaries

are markedly dilated and congested. Round-celled infiltration is occasionally found in the interstitial tissue. Examination of the stomach contents yields characteristic results (see page 95).

Symptoms.—In mild forms of gastric catarrh due to dietetic errors the patients complain of a feeling of weight in the pit of the stomach, followed by a sensation of fulness. Belching affords relief. In some cases there is nausea and in the more severe type of acute gastritis the onset of the disorder is characterized by gastric pains, nausea and vomiting, rise of temperature, loss of appetite, and constipation or diarrhea. The vomited material usually consists of ill-smelling and fermented masses, acid in reaction, its total acidity varies, and as a rule there is either no free hydrochloric acid at all or less than normal. On the other hand, there may be hyperacidity and hypersecretion accompanied by pyrosis. A high total acidity is occasionally caused by the presence of the organic acids—acetic and butyric. Emesis, or retching after the stomach has been emptied, often results in the evacuation of mucobiliary masses. The tongue is coated and the breath fetid. The region over the stomach is sensitive to pressure, and the stomach itself is slightly distended. Acute gastritis may be afebrile, or there may be a temperature of 102° to 104° F.

Course.—The course of acute gastritis depends largely upon the intensity of the attack; its usual duration is from one to three days. An early emesis gives great relief, so that the distressing symptoms often rapidly subside. Sometimes, however, vomiting is followed by lassitude, weakness, and cephalalgia. Acute gastric catarrh may pass from the stomach to the intestine, involving both, so that we have a gastroenteritis. Though patients usually recover from mild attacks in two or three days, the so-called "weak stomach" remains and the patient has more or less prolonged periods of anorexia.

Prophylaxis. Persons subject to attacks of acute gastric catarrh should be on their guard against dietary indiscretions. They should avoid rich food, food that is either too cold or too hot, unripe fruits, and whatever may have been implicated in causing previous attacks. Were patients to avoid such articles of diet and refrain from habits and excesses known to themselves to be causative factors in acute gastric catarrh, the occurrence of this disease could be prevented to a very marked degree.

Treatment. To get rid of the undigested material, the stomach empties itself by vomiting, or by passing the contents on to the small intestine, where they may set up a diarrheal discharge.

When vomiting does not take place from the irritation caused by the mass of undigested food in the stomach, we should lend our assistance to bringing about evacuation of the stomach contents. The best method of cleansing the stomach is by the use of the stomach tube. Since the object is not medication, but simply

mechanical elimination, it is sufficient to wash out the stomach with lukewarm water to which sodium bicarbonate, a teaspoonful to the pint, has been added. This will promote the solution of mucus. I strongly commend the use of the stomach tube for promptness and thoroughness in the evacuation of the stomach; it has the additional advantage that it does not irritate the gastric mucosa as do emetics given by mouth. In performing lavage, the patient should be instructed to assume different positions to facilitate the thorough cleansing of the stomach. Usually a single lavage is sufficient if it be thoroughly done. In children, lavage is the only method of cleansing the stomach that should be considered. In infants and very young children it may be accomplished by means of a Nélaton catheter.

After lavage the retching ceases and the general condition improves. It is evident that gastritis cannot be cured so long as decomposed food remains in the stomach. Emetics proper are reserved for those cases in which, for one or another reason, it is impossible or impracticable to use the stomach tube. The most useful emetic is:

	Gm. or Co.	
R—Antimonii et potassii tartratis	0 05	gr. j
Pulveris radices ipecacuanhæ	1 00	gr. xv
Misce et ft. pulv. no. v.		
Sig.—One powder every quarter of an hour until vomiting occurs.		

The following may be administered to children:

	Gm. or Co.	
R—Pulveris radices ipecacuanhæ	2 0	3ss
Syrupi amygdalæ	20 0	3v
Misce.		
Sig.—One dessertspoonful every ten minutes until vomiting is induced.		

When the administration of emetics by mouth is inadvisable, on account of its tendency to increase the irritable condition of the stomach, the hypodermic injection of apomorphin is useful. Hypodermic tablets of apomorphin ready for use are to be had, and fresh supplies should be carried in the regular medicine case. The action of apomorphin is rapid and certain.

After the stomach has been thoroughly emptied and cleansed, all food should be interdicted for the next twenty-four to forty-eight hours. This edict will not be difficult to enforce, since the patients have little or no appetite. Thirst may be allayed by means of cracked ice. Carbonated waters, iced milk, brandy and soda, and lemonade are acceptable and generally harmless.

Preparations containing menthol quiet and anesthetize the hypersensitive mucosa, acting at the same time as antiseptics:

	Gm. or Co.	
R—Mentholis	1 0	gr. xv
Alcoholis		
Syrupi	33 0	3j
Misce.		
Sig.—One teaspoonful every hour.		

Validol is a good substitute for menthol. It is a preparation of menthol and valerianic acid, containing about 30 per cent. of free menthol. It may be prescribed to be taken three times a day in doses of 0.6 to 1 Cc. (10 to 15 minims).

Bicarbonate of sodium, either alone or with such antizymotics as resorcinol and salicylic acid, may be given, should the contents of the stomach be markedly acid:

	Gm. or Cc.	
R—Resorcinolis	0.6	gr. x
Sodii bicarbonatis		
Bismuthi salicylatis	AA 4.0	3j
Misce et ft. pulv. no. x		
Sig. One powder every two hours.		

The coated tongue may be nicely cleansed by means of a clean piece of soft linen moistened with lemon juice.

A marked degree of pyrosis can be relieved by the following:

	Gm. or Cc.	
R—Magnesii oxidii,		
Sodii bicarbonatis,		
Olei sacchari menthae piperitæ	AA 10.0	5ijss
Misce et ft. pulv.		
Sig. One-quarter of a teaspoonful in water every two hours.		

For the relief of pain:

	Gm. or Cc.	
R—Codeinæ phosphatis	0.12	gr. ij
Aquæ menthae piperitæ	30.0	3j
Misce		
Sig. One teaspoonful twice or three times a day.		

For acid eructations:

	Gm. or Cc.	
R—Resorcinolis	1.0	gr. xv
Aquæ destillatæ,		
Aquæ menthae piperitæ	AA 45.0	3ss
Misce		
Sig. One teaspoonful every two hours.		

Diet. After twenty-four to forty-eight hours' rest, liquid food (no other) should be given—soups and gruel in small but gradually increasing quantities. The yolk of an egg may be added to the soup. Later, this diet may be followed by milk sipped slowly, fowl, minced ham, crackers, eggs, and fillet of beef. This is usually sufficient for an ordinary case of simple acute gastritis. Should the appetite continue poor, it may be stimulated by hydrochloric acid dilute, 1 Cc. (15 minims) before meals, in lemonade or compound tincture of cinchona. Fluidextract of condurango, 1 Cc. (15 minims) three times a day, before meals, has a good effect.

Pain is sometimes complained of, though it is rarely of such intensity as to require treatment. Moderate pains and gastric pressure are best treated by hydragric measures. A Priesnitz bandage, renewed every two or three hours, is of good service

in such cases (see page 250). Should the pains be more severe, moist applications or hot dry compresses, hot bottles or the electric pad are indicated. These appliances may be continued for some time if necessary. The consideration of analgesic and narcotic remedies must be reserved for cases accompanied by excessive pain; these drugs must not, however, be given by mouth, but should be administered in suppository form only. Extract of opium, 0.03 to 0.05 Gm. ($\frac{1}{4}$ to 1 grain); codein phosphate in the same dose; extract of belladonna, 0.03 Gm. ($\frac{1}{4}$ grain)—these, alone or in combination, are the drugs employed. Morphin is apt to induce vomiting.

The intestinal tract may be affected in acute gastric catarrh by the presence of irritating substances from the stomach, so that instead of a simple acute gastritis we have an acute gastro-enteritis. When there is reason to suspect the presence of decomposed and irritating masses in the intestine, it is good treatment to induce evacuation. Calomel is the best remedy we have for this purpose. It is an excellent remedial agent in the treatment of gastro-intestinal disorders of children. The dose for adults is 0.12 Gm. (2 grains) twice a day, or 0.01 Gm. ($\frac{1}{4}$ grain) every hour for ten doses. Castor oil is also a useful evacuant. Patients who cannot take castor oil will readily take Rochelle salt, 4 Gm. (3j) in a half-glass of water; it should be taken in the morning when the stomach is empty. After thorough evacuation of the bowels, three or four days may be allowed to elapse before the next movement. Should constipation persist, an enema of warm water, plain or containing soap, oil, glycerin, vinegar, soda or cottonseed oil, should be given. After cleansing the intestine, such intestinal antiseptics as resorcinol or salicylate of bismuth may be considered.

ACUTE INFECTIOUS GASTRITIS.

Gastric catarrh may occasionally assume what is known as the grave form.

Etiology. Usually the exciting cause of infectious gastric catarrh consists of microorganisms introduced into the stomach with articles of food, decomposed meat or fruit, or food or drink which may not appear to be tainted, such as impure milk, or water from infected wells. The grave form of acute gastric catarrh may thus become epidemic. It is often a very difficult matter to determine with absolute certainty the cause of this disease.

Pathology.—The pathologic changes accompanying all forms of acute gastritis are similar to those of the mild form; the difference is one of degree only. There is marked hyperemia, tumefaction and reddening of the gastric mucosa, in which the glandular epithelium and interstitial tissue participate. There is also emigration of wandering cells from the blood—leukocytes, more or less

fibrin—which, in addition to the acute edema of simple "gastritis," gives the histologic picture of an essential inflammation. Frequently there are small petechial hemorrhages.

Symptoms.—The symptoms described in the section on Acute Gastric Catarrh are present here in aggravated form, consisting of violent pains accompanied by persistent and severe vomiting and marked prostration. Fever, usually absent in mild gastric catarrh, is a constant symptom of the grave variety. In fact, the febrile disturbance is a fairly reliable index of the gravity of the disease. These severer forms of acute gastritis are sometimes due to dietary indiscretions; but they are more frequently the result of infection, so that this form of the disease is designated acute infectious gastric catarrh.

The fever (gastric fever) is of marked intensity and of the continued or remittent type. Other symptoms are: violent throbbing headache, insomnia, thirst, rapid pulse, and occasional delirium. In the febrile cases there is a marked diminution of acid secretion; the fever itself in all probability reduces the secretion of hydrochloric acid. The disease ordinarily runs from ten to fourteen days; in some instances the fever may persist for three weeks. In very old and very young patients this form of gastritis may assume an alarming character.

The severe forms of infectious gastritis exhibit clinical symptoms similar to those that are caused by the introduction into the stomach of organic poisons, such as the metabolic products of infectious microorganisms—toxins and ptomaines. The course of the disease is usually, but not always, severe.

Treatment.—The treatment of these severe forms of acute gastric catarrh is based upon the same principles as that of the milder forms. The stomach must be emptied and cleansed as quickly and thoroughly as possible by means of lavage. When the disease is due to infection it is well to wash out the stomach with antiseptic solutions; for example, salicylic acid 1 to 2 parts in 1000 of water, or dilute boric acid solution (3:1000 to 5:1000). Emetics should not be employed if it is possible to empty the stomach in any other way. Food should be interdicted for a number of days in the case of robust patients, to give the stomach needed rest. Thirst and persistent vomiting are to be met by small doses of cold mineral waters, carbonated waters either with or without fruit juices, cracked ice, or cold tea. The general condition of the patient, his pulse and temperature, must be constantly under observation. Wine, brandy, cognac, champagne, Tokay wine and strong coffee are to be administered to the aged and weak as indicated.

When a patient is in a condition to partake of food, particular care should be exercised in regard to the kind and quantity permitted. At first only liquid foods, such as bouillon with yolk of

egg, meat juices and extracts, albumin water and leguminous soups, should be allowed. Should obstinate vomiting interfere with eating, nutrient enemata may be given. Great caution should be exercised when the patient is passing from liquid to solid food. The initial solids should consist of sweetbread, brain, boiled fowl (chicken, squab), minced raw meat, minced ham, meat jelly, flour and milk gruel, tapioca, mashed potatoes, milk, crackers, or zwieback. The return to a full diet should be very gradual, not complete until ten to fourteen days after the cessation of all the symptoms.

Medicinal Treatment. The same drugs prescribed in the treatment of milder gastric catarrh are indicated in the infectious forms. Since in these severe acute cases the hydrochloric acid secretion is diminished, dilute hydrochloric acid well diluted with water should be given three times a day (see page 258). This will serve the additional purpose of allaying the thirst. Resorcinol may be given for nausea and foul-smelling eructations. To reduce fever, 0.3 Gm. (5 grains) of quinin or acetphenetidin may be given; or recourse may be had to the tepid or cold bath. Calomel, 0.015 Gm. ($\frac{1}{2}$ grain) three times a day, will often exert a good influence on the course of the disease.

When the infection has passed to the intestine, calomel should be given, to be followed if necessary by resorcinol with salicylate of bismuth; the following formula will be found useful:

	Gm. or Gs.	gr. xlv
R—Bismuthi salicylatis	3 0	gr. xlv
Resorcinolis	2 0	gr. xxx
Glycerini	15 0	3 ss
Aquæ	200 0	3 viij
Misce		
Sig.—One tablespoonful every three hours.		

TOXIC GASTRITIS.

Etiology.—Severe toxic gastritis may be caused by chemical poisons, such as concentrated mineral acids, caustic alkalis, ammonia, phenol, oxalic acid, alcohol, phosphorus, arsenic, potassium cyanid, potassium chlorate, corrosive sublimate, lysol, and others.

Pathology.—The essential feature of intoxication by the heavy metals and phenol is that at an early stage no histologic change is seen, even though the tissue be dead. It is "fixed" just as is tissue in a test-tube. At this stage there is no reaction in this part of the stomach wall. Later, inflammatory reaction sets in. The most marked alterations of the gastric mucous membrane are produced by the corrosive poisons, acids and alkalis, oxalic acid, phenol, lysol, and corrosive sublimate. At first the wall of the lower end of the greater curvature not far from the pylorus, or the posterior wall of the stomach, is attacked by these poisons, the location depending

on the position of the patient (that is, whether lying or standing) when the poisonous substance is ingested. The gastric mucosa is hyperemic and greatly swollen, subsequently becoming ulcerated; the ulcers sometimes penetrate to the serous coat, or even to complete perforation. In recovery the patient may have pronounced disturbance of the motor and chemical functions of the stomach; there is apt to be an alteration in the shape and size of the organ, or esophageal stricture due to cicatrization. Alcohol and phosphorus do not produce such marked lesions, but cause an intense irritation and inflammation of the mucosa together with fatty degeneration of the glandular epithelium.

Symptoms.—The symptoms will vary according to the amount of poison taken. There is always intense burning pain in the pharynx, along the esophagus, and especially in the stomach. Vomiting soon commences, but does not bring relief to the patient. The vomited matter contains an admixture of blood. The stomach is usually distended and the abdomen exceedingly sensitive to pressure. Thirst is always a feature. In cases of great severity the pulse is small, the lips blue, and there is perspiration, with slight coma; death may occur in collapse.

Prognosis.—The prognosis in such cases depends upon the quantity of poison taken, as well as upon the condition in which the patient is found. Every case of poisoning should be considered serious, and recovery a matter of doubt.

Treatment.—The prime requirement is to remove the poison from the stomach with the utmost speed, and this is best accomplished by lavage. It is sometimes dangerous to attempt to introduce the tube, owing to the possibility of perforation. Especially is this likely to happen in poisoning by acids or caustic alkalis. In all such cases the best mode of treatment is to effect a dilution of the poison, and if possible its neutralization.

In the treatment of poisoning by inorganic acids, alkalis are indicated to neutralize any free acid in the pharynx, esophagus or stomach. Large doses of magnesium oxid, 200 Gm. (℥viij) in four parts of water; sodium carbonate diluted in a mucilaginous vehicle; lime-water, powdered chalk, and large quantities of sodium bicarbonate, are suitable antidotes. Care should be exercised in the employment of chalk and bicarbonate of sodium, owing to the generation of carbon dioxid on contact with the acid. In poisoning by organic acids, saccharated lime may be given, in addition to the other substances mentioned, for the purpose of converting the acid into a nearly insoluble lime salt. Cracked ice should be administered, and ice packs applied over the region of the stomach. Morphin may be given for the relief of pain.

In cases of poisoning by alkalis, such acids as acetic or citric are indicated to neutralize the caustic effect of the poison. Lysol and phenol poisoning call for thorough lavage with large quanti-

ties of water (2 or 3 liters); large doses of sodium sulphate are useful; lime-water and saccharated lime produce the comparatively harmless phenolate of lime. It is well to note, too, that grain alcohol is the nearest approach we have to an ideal drug for neutralizing the effect of phenol. In phosphorus poisoning the treatment consists of long-continued lavage and the subsequent administration of half a teaspoonful of turpentine every half-hour (see page 357).

PHLEGMONOUS GASTRITIS.

This is among the rarest of gastric diseases. The earliest description of the disease would seem to be in a communication by Veranadeus in 1620. In the latter half of the seventeenth century and in the beginning of the eighteenth there were published observations on phlegmonous gastritis by Borel (1656), Sand (1701), Vorwaltner, and Bonet. These observations describe the circumscribed form only. Andral (1839) and Cruveilhier appear to have been the first to observe the diffuse form of purulent infiltration of the gastric walls. In their case a fortunate accident led to incisions into the stomach walls, which revealed a diffuse submucous suppurative inflammation. Since 1860 many papers on both the circumscribed and the diffuse form have been published, and a very admirable monograph by Leith, of Edinburgh, in 1896. Leith was able to collect only 51 positive cases of the diffuse form of phlegmonous gastritis in the entire literature of the subject, and the total number of cases of both diffuse and circumscribed forms is given as 85.

The disease is characterized by a purulent inflammation of the walls of the stomach, originating in the submucous coat and gradually extending to the other layers. A primary and a secondary or metastatic form of the disease have been distinguished. The condition has been classified also as "diffuse phlegmonous gastritis," in which the purulent infiltration of the stomach extends over a large area; and "circumscribed," or so-called abscess of the stomach. It usually runs an acute, though occasionally a sub-acute, course. Only about 90 cases have been reported, of which number the majority were males. The metastatic form of the disease usually originates in infectious diseases—puerperal fever or pyemia.

Etiology.—The cause of the primary affection is obscure. Alcoholism has been suggested. Traumatism, dietetic errors, exposure, food and drug poisoning, puerperal fever, and carcinoma have been noted as contributory factors; they undoubtedly lessen the power of resistance so that the stomach more easily becomes a nidus for pyogenic bacteria. Kinnicutt reports a case of phlegmonous gastritis in which bacteriologic examination revealed the universal presence of the streptococcus; it was most abundant

in the connective tissue of the submucosa and the muscularis. Two cases were reported by Robertson, in which the direct cause was a virulent streptococcus in the gastric submucosa, entering through a defect in the mucosa or carried by the blood or lymph currents. If the disease be due to bacterial infection of the submucous coat through some small abrasion of the mucosa—which is the most plausible explanation—then it is strictly analogous to cellulitis of the subcutaneous tissues due to a cutaneous defect healed long before the cellulitis is observed. This hypothesis gives a clue to the surgical treatment, to be discussed later.

The secondary form of this disease, apparently due to metastatic infection, may originate from carcinoma or ulcer of the stomach. In one case gastritis phlegmonosa has been observed to follow an enterostomy.

Pathology. The essential lesion is a widespread inflammatory change in the submucous coat, which is greatly thickened, usually of a yellowish-white color, and so much softened that it resembles pus. Microscopically the appearance is that of fibrin with masses of leukocytes entangled in it. This change is nearly always more marked in the pyloric half of the stomach, a fact which may bear some relation to the anatomic situation of the oxyntic or acid-producing cells of the gastric mucosa. The muscular coat shows varying degrees of infection and degeneration of the muscular elements. The serous coat is sometimes unaffected, but it may show leukocytic infiltration, especially in cases where a secondary purulent peritonitis is present. The mucosa is in many instances normal, but in others it is acutely inflamed—raised from its bed in ridges; in others again the deeper layers of glandular tubules are atrophied; while in a few of the recorded cases the mucous surfaces have been pitted with tiny apertures, giving it the appearance of a sieve, through the meshes of which pus could be squeezed from the infiltrated submucous layer. The duodenum is very rarely altered, and then only the mucosa is inflamed. Of secondary lesions, peritonitis, seropurulent or purulent, is the commonest, and it has been found in rather more than half the cases. Pericarditis, pleurisy, and abscess of the liver have also been observed.

Symptoms and Course. The course of the diffuse form of this disease is atypical. We may have a severe acute gastritis, with high fever, sometimes as high as 104° F., violent pains and uncontrollable vomiting, the abdomen greatly distended, the pulse feeble, symptoms of collapse follow and the termination is usually fatal.

The course of the circumscribed form is similar, except that it is of longer duration, extending sometimes over several weeks. Sometimes a tumor can be felt in the region of the stomach.

The prognosis in both forms of phlegmonous gastritis is very grave, practically hopeless. Up to 1896, Leith, who had pub-

lished the best account of the disease, found no authentic cases of recovery; and from the records of cases since that date it appears probable that the few recoveries noted were really not cases of primary phlegmonous gastritis, but abscess of the stomach.

Treatment. Since the diagnosis is never positive, the treatment must be more or less symptomatic. There is no successful internal treatment of this disease. The stomach should be spared as much as possible. Food and drink should be administered per rectum. Ice-bags, and cocaine, morphin or codein hypodermically, are indicated for the relief of pain. High temperatures are to be controlled by the use of antipyretic drugs. Stimulants should be administered early in the disease, as well as when symptoms of collapse appear. If the nature of the infecting organism can be learned, the appropriate bacterial vaccine should be administered.

This disease is essentially surgical. Gastrostomy or gastroenterostomy is suggested as an appropriate method of dealing with the lesion surgically, but it is difficult to see how a surgical operation is going to cure a cellulitis of the stomach wall. Probably incisions down to the submucous coat, with free exposure of the stomach wall, and packing-off of the peritoneal cavity with gauze left in position for several days, would offer the best chance of recovery. This is the treatment of cellulitis in subcutaneous lesions, and if it could be effected without infecting the general peritoneal cavity it seems reasonable to hope that satisfactory results might be obtained.

CHAPTER XXIII.

CHRONIC GASTRITIS—ACID GASTRITIS—SUBACID GASTRITIS—ANACID GASTRITIS; ACHYLIA GASTRICA.

CHRONIC GASTRITIS.

CHRONIC catarrhal gastritis is a chronic inflammation of the gastric mucous membrane, of varying degrees of intensity, presenting symptoms more or less characteristic of widely different forms of gastric derangement. It is a disease which requires for its positive diagnosis and, as a rule, for even probable diagnosis, an examination of the gastric secretion.

Etiology.—When the irritating cause of acute gastritis persists, chronic gastritis is the natural consequence; but there are generally other, often altogether different, etiologic factors. Gastritis occurs as a concomitant manifestation in many grave affections of the stomach, and we find it almost always in carcinoma leading to atrophy of the mucous membrane. In all conditions which induce stagnation in the area of the portal vein, notably cardiac and pulmonary affections, chronic gastritis supervenes as a result of the engorgement. It also occurs in conditions which disturb the composition of the blood, such as chronic intoxications, chlorosis, anemia, chronic renal affections, gout, diabetes, and infectious diseases. After grave acute infections, inflammatory changes of the gastric mucosa are easily demonstrable.

Extreme temperatures, both hot and cold, attacking the gastric mucosa, may in the course of time cause permanent degenerative changes. Overloading of the stomach, food that is hard to digest, insufficient mastication and defective teeth are very important factors. Neglect in the care of the mouth, especially when artificial teeth are worn, may lead to gastric catarrh. It often happens that gastric symptoms occur in perfectly healthy individuals shortly after they commence to use artificial teeth which they do not keep clean. Oral sepsis is the cause of many cases of chronic gastritis. Affections of the nose, the nasopharynx, the accessory sinuses, or any ulceration, may lead to gastritis if the pathologic secretions are swallowed (see page 290).

In the etiology far too little attention is given to the use, or rather abuse, of articles of diet, except perhaps alcohol, which has long been considered one of the causes of chronic gastritis. Increased importance is now also given to tobacco as a causative

factor. Such medicaments as balsam copaiba, menthol and santal oil may occasionally cause chronic gastritis.

Pathology.—In chronic gastritis it is not the superficial epithelium alone that is affected, but the inflammatory process extends to the glandular epithelium and to the interstitial tissue. In the initial stage of simple chronic gastritis the mucous membrane is pale gray in color and covered with closely adherent tenacious mucus. The veins are enlarged, and patches of ecchymosis are sometimes seen. The glands are subject to parenchymatous and interstitial inflammation, presenting a microscopic picture of erosion, cloudy swelling, or atrophy, depending upon the stage of the disease. It is not possible to differentiate between the peptic and the parietal cells, owing to the fact that the tubes have lost their regular form and instead we have, as Ewald expresses it, "atypical branching like the fingers of a glove." There is an infiltration of round cells and proliferation of connective tissue, which exert pressure on the glands, thus inhibiting their normal functions. As these pathologic changes become more marked the secretion becomes progressively less until the atrophic stage is reached and secretion ceases entirely. Meanwhile a mucoid degeneration of the cells lining the tubules takes place and may even extend to the fundus of the glands. There is a proliferation of connective tissue, so that, toward the pylorus in particular, the stomach wall has a rough, wrinkled, mammillated appearance, the *état mamelonné* of the French, a condition which is sometimes so prominent that it has been described as *gastritis polyposa*. The pathologic changes may even lead to stenosis of the pylorus. One of the important features of the fibrosis which develops between the different layers of the stomach wall (especially in the submucosa) is that it interferes with motility or elasticity of the mucosa upon the muscularis. It is therefore more or less essentially associated with variation of motility.

The inflammation in the more aggravated cases may pass to the muscular layers, causing partial destruction, to be replaced by connective-tissue fibers. Belonging to this form of the disease is sclerotic gastritis (*cirrhosis ventriculi*), in which the walls of the stomach undergo a connective-tissue metamorphosis, sometimes to such an extent that the stomach is greatly reduced in size. The fact is now recognized that there may be such destruction of the glandular elements by a progressive growth of interstitial tissue that ultimately scarcely a trace of secreting tissue remains.

Symptoms.—The local symptoms have a strong resemblance to those of other forms of gastric disturbance. The disease, as a rule, develops very slowly, and, as in the case of most chronic diseases, changes from time to time. The appetite varies; sometimes it is very poor, and sometimes it is good. Patients usually complain of a disagreeable taste, which they describe as salty or

pappy, or at times sour; of thirst, salivation, and eructation of gas or food remnants, which may be sour, rancid, or tasteless. The breath is often fetid. Nausea is rather common. Pressure and fulness are experienced after eating. Patients complain of palpitation of the heart. Belching (which is very annoying to the patient) relieves both the pressure and the consequent palpitation. Irregular stools, constipation and diarrhea are commonly met with in chronic catarrhal gastritis. Patients suffer from headaches, vertigo, and disturbed sleep. There are vasomotor disturbances, with sensations of coldness of the extremities.

Objective Symptoms. The patients, as a rule, appear to be well nourished; but some are seen to have lost weight and look emaciated. The tongue is usually coated gray or yellowish-gray; still, in many cases of well-marked chronic gastritis the tongue is clean. There may be no offensive odor in the mouth, or if there is any it may be due to carious teeth or some pathologic condition of the nose or throat. The gastric region often appears bloated. Palpation reveals slight sensitiveness of the entire area over the stomach. The pylorus may be palpated when thickened by muscular hypertrophy.

Diagnosis.—It is seen that there are no symptoms, either subjective or objective, which are pathognomonic of the disease. An approximate diagnosis can be established only by an examination of the secretory and motor functions of the stomach and the anamnesis. The presence of mucus in the stomach must be ascertained before we are justified in making a diagnosis of chronic gastric catarrh. From the stomach under normal conditions it is possible to obtain only a few isolated flakes of mucus, even after most thorough lavage. In chronic gastritis mucus is usually present in marked quantities, and is found mixed with food remnants. Mucus which enters the stomach from the nose, pharynx or trachea is found upon the surface of the liquid or food removed from the stomach with the stomach tube. In chronic gastritis the picture of mucus entangling large numbers of cells is characteristic, but free polynuclears are of greater significance.

The acid secretion in chronic gastric catarrh varies. In the initial stages the percentage of free hydrochloric acid is often found to be normal. Sometimes there is hyperacidity, a condition which corresponds to the *acid gastritis* described by Boas. It is a gastritis with increased production of mucus and an abnormally active secretion of hydrochloric acid. The mucus siphoned out early in the morning after the night's fast may give a positive hydrochloric acid reaction. Acid gastritis is an early form of chronic gastritis, and is found very frequently in alcoholic patients. As the disease progresses, the secretory function becomes impaired, with a resultant decline from the normal amount of free hydrochloric acid. In the more protracted cases there is no free hydro-

chloric acid at all, and the other constituents of the gastric secretion are very much diminished in quantity. The diminution of free hydrochloric acid is in direct proportion to the intensity of the disease process; when very marked, the condition is designated *subacid gastritis*. Further progress of the disease converts the subacidity into anacidity, a condition in which the formation of pepsin begins to fail. This pathologic state is known as *anacid gastritis*.

The final stage of the disease is that of chronic anacid gastritis with atrophy of the secreting glands. The pathologic changes in the gastric mucosa are so great as to preclude the possibility of restitution of the secretory functions of the stomach. When this stage is reached, rennin as well as pepsin and hydrochloric acid are absent. The term "subacidity" is applied to cases in which free hydrochloric acid is decreased and the total acidity is less than 40 degrees; the secretion of the ferments may be normal (see page 95).

It is a question whether gastric anacidity may exist as an independent primary affection (*achylia gastrica*), or whether it is to be always regarded as a later stage of chronic gastritis characterized by atrophy of the gastric mucosa. The possibility of gastric anacidity as a primary affection would seem to be assured by cases of purely nervous or functional disturbance, or by cases in which there is an inherent deficiency in the secretory function of the stomach. The examination of stomach contents in such cases reveals an unaltered condition of the test meal. The particles of bread are larger or smaller, depending upon the thoroughness of mastication, and have the appearance of being merely moistened or softened by the water. Mucus is not present. The total acidity is extremely low—4 to 6 degrees, and frequently zero. The reaction of the stomach contents is very slightly acid, sometimes amphoteric on account of the presence of phosphates in the food.

Very often individuals with anacidity or subacidity feel perfectly well, or at least experience no great discomfort. They appear well nourished. Cases have been observed in which gastric anacidity had persisted for periods of twelve to fifteen years. In this class of cases the power of the stomach to digest protein is entirely absent, so that the small intestine receives all the protein in an unchanged condition. The test-diet stool is characteristic under such conditions (see Chapter IV).

The motor activity of the stomach is usually normal. Stagnation is found only in those cases in which there is hypertrophy of the muscular layers of the pylorus.

Prognosis.—The prognosis of chronic gastritis is favorable; the disease responds to treatment, so that a complete cure or material improvement may be anticipated. Relapses are, however, likely to occur.

pappy, or at times sour; of thirst, salivation, and eructation of gas or food remnants, which may be sour, rancid, or tasteless. The breath is often fetid. Nausea is rather common. Pressure and fulness are experienced after eating. Patients complain of palpitation of the heart. Belching (which is very annoying to the patient) relieves both the pressure and the consequent palpitation. Irregular stools, constipation and diarrhea are commonly met with in chronic catarrhal gastritis. Patients suffer from headaches, vertigo, and disturbed sleep. There are vasomotor disturbances, with sensations of coldness of the extremities.

Objective Symptoms. The patients, as a rule, appear to be well nourished; but some are seen to have lost weight and look emaciated. The tongue is usually coated gray or yellowish-gray; still, in many cases of well-marked chronic gastritis the tongue is clean. There may be no offensive odor in the mouth, or if there is any it may be due to carious teeth or some pathologic condition of the nose or throat. The gastric region often appears bloated. Palpation reveals slight sensitiveness of the entire area over the stomach. The pylorus may be palpated when thickened by muscular hypertrophy.

Diagnosis. It is seen that there are no symptoms, either subjective or objective, which are pathognomonic of the disease. An approximate diagnosis can be established only by an examination of the secretory and motor functions of the stomach and the anamnesis. The presence of mucus in the stomach must be ascertained before we are justified in making a diagnosis of chronic gastric catarrh. From the stomach under normal conditions it is possible to obtain only a few isolated flakes of mucus, even after most thorough lavage. In chronic gastritis mucus is usually present in marked quantities, and is found mixed with food remnants. Mucus which enters the stomach from the nose, pharynx or trachea is found upon the surface of the liquid or food removed from the stomach with the stomach tube. In chronic gastritis the picture of mucus entangling large numbers of cells is characteristic, but free polynuclears are of greater significance.

The acid secretion in chronic gastric catarrh varies. In the initial stages the percentage of free hydrochloric acid is often found to be normal. Sometimes there is hyperacidity, a condition which corresponds to the *acid gastritis* described by Bous. It is a gastritis with increased production of mucus and an abnormally active secretion of hydrochloric acid. The mucus siphoned out early in the morning after the night's fast may give a positive hydrochloric acid reaction. Acid gastritis is an early form of chronic gastritis, and is found very frequently in alcoholic patients. As the disease progresses, the secretory function becomes impaired, with a resultant decline from the normal amount of free hydrochloric acid. In the more protracted cases there is no free hydro-

chloric acid at all, and the other constituents of the gastric secretion are very much diminished in quantity. The diminution of free hydrochloric acid is in direct proportion to the intensity of the disease process; when very marked, the condition is designated *subacid gastritis*. Further progress of the disease converts the subacidity into anacidity, a condition in which the formation of pepsin begins to fail. This pathologic state is known as *anacid gastritis*.

The final stage of the disease is that of chronic anacid gastritis with atrophy of the secreting glands. The pathologic changes in the gastric mucosa are so great as to preclude the possibility of restitution of the secretory functions of the stomach. When this stage is reached, rennin as well as pepsin and hydrochloric acid are absent. The term "subacidity" is applied to cases in which free hydrochloric acid is decreased and the total acidity is less than 40 degrees; the secretion of the ferments may be normal (see page 95).

It is a question whether gastric anacidity may exist as an independent primary affection (*achylia gastrica*), or whether it is to be always regarded as a later stage of chronic gastritis characterized by atrophy of the gastric mucosa. The possibility of gastric anacidity as a primary affection would seem to be assured by cases of purely nervous or functional disturbance, or by cases in which there is an inherent deficiency in the secretory function of the stomach. The examination of stomach contents in such cases reveals an unaltered condition of the test meal. The particles of bread are larger or smaller, depending upon the thoroughness of mastication, and have the appearance of being merely moistened or softened by the water. Mucus is not present. The total acidity is extremely low—4 to 6 degrees, and frequently zero. The reaction of the stomach contents is very slightly acid, sometimes amphoteric on account of the presence of phosphates in the food.

Very often individuals with anacidity or subacidity feel perfectly well, or at least experience no great discomfort. They appear well nourished. Cases have been observed in which gastric anacidity had persisted for periods of twelve to fifteen years. In this class of cases the power of the stomach to digest protein is entirely absent, so that the small intestine receives all the protein in an unchanged condition. The test-diet stool is characteristic under such conditions (see Chapter IV).

The motor activity of the stomach is usually normal. Stagnation is found only in those cases in which there is hypertrophy of the muscular layers of the pylorus.

Prognosis. The prognosis of chronic gastritis is favorable; the disease responds to treatment, so that a complete cure or material improvement may be anticipated. Relapses are, however, likely to occur.

ACHYLIA GASTRICA.

"Achyilia gastrica" is a term introduced into medical literature by Einhorn to denote absence of gastric secretion. The stomach contents contain no free or combined hydrochloric acid; the ferments are likewise absent or greatly reduced in amount. Achyilia is a sign of disturbed function of the stomach which may accompany such diseases as carcinoma, severe anemia, or chronic gastric catarrh. It may also occur as a purely functional disturbance wholly apart from primary organic disease of the stomach or other organs.

In achyilia gastrica there is no chymification of the gastric contents; the aspirated parts of a test breakfast have the appearance of crumbs of bread in water (see Chapter II). The contents of the stomach are expelled with abnormal rapidity. A test breakfast may be emptied into the duodenum in a quarter to half the normal time (hypermotility). There being no hydrochloric acid, the pylorus does not close as it would under normal conditions (see page 94).

Achyilia gastrica senilis is a true wasting disease and is found to consist of a senile atrophy of the gastric mucosa. Achyilia following chronic atrophic gastritis occurs between the ages of thirty and fifty. In these cases, as distinguished from the cases of primary achyilia, there is more or less mucus mixed with the stomach contents.

Achlorhydria hemorrhagica gastrica is a condition of achyilia due to reflex inhibition, characterized pathologically by superficial ulceration of the mucosa, hyperemia and extravasation of blood. Etiologically it is often secondary to chronic appendicitis, cholecystitis, or pancreatitis (see page 427). In a majority of cases diplococci and streptococci are found in the gastric mucosa. Their presence leads us to believe that the condition may be of focal origin (see page 290). The diagnosis is made by the finding of an achlorhydria and the demonstration of the presence of occult blood (see page 86). The treatment is the same as that of chronic gastritis, with removal of the original cause by surgical intervention.

Carcinoma of the stomach is preceded or attended by achyilia. When achyilia occurs after the age of forty, the possibility of carcinoma should be borne in mind. Every case of carcinoma of the fundus at some period of its existence shows no other signs, subjective or objective, than those of chronic gastritis and achyilia gastrica.

In cases of chronic cholelithiasis, and particularly pancreatic affections, the possibility of achyilia gastrica being the cause should not be overlooked; nor should we forget the frequent etiologic rôle of this gastric condition in intermittent intestinal catarrhs (colitis, gastrogenic diarrhea) continuing for days or weeks and alternating with normal evacuation or with constipation.

Etiology.—The etiology of achylia gastrica is still unexplained. From clinical observation it would seem that the internal secretions are in some way involved. Then, again, the quick return to normal gastric secretion after an appendectomy for chronic appendicitis associated with achylia gastrica would suggest an impingement on the nerve terminals, with reflex inhibition of gastric secretion. Often patients do not consult a physician until the onset of a secondary diarrhea or pancreatic insufficiency. They may give a personal history of no previous symptoms. Even the diarrhea (see Chapter XXXVIII) may have existed for years without causing any special complaint, being a deuteropathic manifestation of increased decomposition and deficient chymification of the ingested food.

Achylia gastrica is always present in pernicious anemia, both conditions being probably due to total atrophy of the gastrointestinal mucosa, which permits the blood to form hemolysins that result in blood degeneration.

Congenital achylia gastrica is caused by a defective primary development of the gastric mucosa, while atrophy is always the result of inflammation. Achylia gastrica often occurs at as early a period as childhood, when a preceding chronic gastritis can be excluded and gastric symptoms have never manifested themselves. These children are taken to the physician on account of defective physical development, debility, anemia, anorexia, and occasional diarrhea. Adults with periodically intermittent diarrhea have often suffered since childhood. They remain emaciated for life, despite all kinds of treatment, even hyperalimentation. After a period of well-being their metabolism is disturbed by slight dietary errors, overexertion, or excitement, leading to diarrhea with its debilitating sequelæ.

This condition is often found as a family affection, most of the sufferers having a pronounced neurasthenic appearance. They differ, however, from ordinary neurasthenic patients by the anacid condition of their stomach contents and by other symptoms of achylia gastrica. A marked feature in neurotic patients is the fact that achylia suddenly occurs and then again disappears (*heteroachylia*); this is explained by the supposition of a temporarily exhausted function of the gastric mucosa. From this consideration it follows that not every case of achylia gastrica implies an advanced, incurable affection of the glands. Derangement of the vegetative nervous system may be the cause of the achylia (see page 387).

Pathology.—The mucous membrane has been for the most part normal in many cases examined, while in some cases the gastric glands were found to be atrophied.

Symptoms.—The clinical symptoms of achylia gastrica resemble those of chronic gastritis: loss of appetite, nausea, vomiting, slight

pains, and eructations. In many instances the patient feels well, and the existence of the disease is discovered by accident.

Achylia gastrica may be unmarked by the presence of any distressing symptoms, or, if such symptoms are present, they may be wholly non-characteristic of the pathologic condition. The symptoms usually consist of diminished desire for food, pressure, fulness in the stomach, discomfort after eating, or eructations. There is often an acceleration of the motility of the stomach, said to be due to the absence of hydrochloric acid; hydrochloric acid, if present, would cause a periodic closure of the pylorus. The food passes with more than normal rapidity into the small intestine. Patients with achylia gastrica may maintain a fair state of health so long as the small intestine is functionally active. Should intestinal digestion, however, become impaired, the result would be a diarrhea (gastrogenic), causing marked emaciation or even endangering life. (See Chapter XXXVIII.)

Cases described as secondary achylia are sometimes found accompanying such diseases as diabetes, tuberculosis, cirrhosis of the liver, cardiac disease, and arteriosclerosis of the abdominal vessels. Then there is that form of achylia which accompanies grave cases of anemia, pernicious anemia, and the anemia due to the *Bothriocephalus latus*. The relation between achylia and these pathologic conditions is not clear. The test-diet stool findings are important (see page 131).

TREATMENT OF CHRONIC GASTRITIS AND ACHYLIA GASTRICA.

Chronic gastritis and achylia gastrica have as a common manifestation a perversion of gastric secretion which consists for the most part of a diminution in activity of the secretory function. This common functional derangement renders it advantageous to discuss the treatment of the two conditions together.

Since repeated attacks of simple acute gastritis may result in the development of chronic gastritis, it is important that the patient avoid any excesses or practices which predispose to the attacks. The food, neither too hot nor extremely cold, and not highly spiced, should be masticated thoroughly, and the patient should avoid overindulgence in alcohol and tobacco. The mouth and teeth must be kept in good condition. Slow eating followed by rest, exercise in the open air, sleeping with the windows open, and cold salt-water sponging at night followed by a brisk rub, are excellent by way of prophylaxis.

Diet.—The regulation of diet is perhaps the most important factor in the treatment of conditions marked by subacidity or anacidity, since a restoration of the secretory functions of the stomach to normal is sometimes impossible.

The power to digest protein is either greatly impaired or altogether absent. The digestion of carbohydrates in the stomach would be satisfactory were it not for the cellulose enveloping the starch granule; but proteolysis must be carried on for the most part or altogether by the small intestine. The unusual demand made upon the small intestine will sooner or later result in impairment of its function. It is seen, then, that rational treatment must be directed toward protecting the stomach and small intestine. A diet rich in carbohydrate, with a minimum of protein, is indicated. The individual tastes of patients should not, however, be ignored. Some patients object to a monotonous diet. To avoid aversion certain concessions may be granted, but all food should be tender and susceptible of thorough mastication. In chronic gastritis spiced foods may be permitted, owing to their stimulating influence upon the appetite. A reduction of the chlorin supply to the body induces a decrease in the quantity and acidity of the gastric juice. The giving of sodium chlorid with food under such conditions assists in remedying the decrease of hydrochloric acid in the gastric juice. In spite of apparent restriction, the choice of appropriate articles of diet may be sufficiently varied. The patient may be allowed all the tender meats, such as fowl, brain, or lean fish. Meat should be thoroughly roasted or boiled; but raw, pickled, or smoked meats and salted fish should be avoided. The daily quantity of meat should not exceed 150 grams (5 ounces), and in severe cases not more than 100 grams (3 ounces) should be taken during the day. Meat may be replaced by eggs, soft-boiled or in the form of egg soups or light omelets. Milk is, as a rule, well borne, and is strongly recommended; it may be employed as a vehicle for somatose or plasmon, and its digestibility may be further increased by the addition of pepsin. Fats in the form of butter and cream are permitted this class of patients. Vegetables may be prescribed in the form of thick strained soups (rice, tapioca, sago, peas, lentils); and mashed potatoes in moderate quantities are permissible. Biscuits, zwieback, toast, stale white bread, which can be broken up fine in the mouth or softened by being dipped into fluids, are indicated. Such condiments as salt, pepper and mustard have a stimulating effect, though they will not bring back the gastric secretion. The meat extracts have a similar action. Pure water or weak lemonade is the most satisfactory beverage for allaying the thirst, and is best taken during the intervals between meals. Carbonated waters are also good.

Gastric motility is usually normal in chronic gastritis. When, however, there is any disturbance in motility, it may be overcome by making the meals small and frequent, thus avoiding the overdistention of the stomach which large meals are apt to induce. The quantity of liquids should be restricted, inasmuch as they tend to produce hyperdistention.

Ewald's diet for chronic gastritis is as follows:

8 A.M.	150 to 200 Gm. tea (1 cup), with 75 to 100 Gm. [3 ozs.] stale white bread, toast or zwieback.
10 A.M.	50 Gm. [2 ozs.] white bread, 10 Gm. butter, 50 Gm. [2 ozs.] cold meat or ham, one-third liter [1 glass] of milk.
2 P.M.	150 to 200 Gm. [1 cup] water, milk, or bouillon of the white meats, 100 to 125 Gm. [3 to 4 ozs.] meat or fish, 80 to 100 Gm. [about 3 ozs.] vegetables, 80 Gm. stewed fruit [one "helping"].
4 to 5 P.M.	One-fourth to one-third liter [1 glass] of warm milk (occasionally mixed with cocoa or coffee).
7 to 8 P.M.	200 Gm. [1 cup] soup or pap, 50 Gm. [2 ozs.] white bread, 10 Gm. butter.
Occasionally at 10 P.M.	50 Gm. [2 ozs.] white bread (biscuits or zwieback), one cup of tea.

The distribution of meals should conform as nearly as possible to the custom of the community in which the patient lives. In order to permit the patient to have a greater variety of food, it is best not to point out a few articles he should eat, but to mention only those he should avoid. Forbid meat with very tough fibers, meat from too old animals, too fresh meat (right after slaughtering), and meat that contains too much fat (like pork); forbid sausages, lobster, salmon, chicken salad, mayonnaise, cucumbers, pickles, cabbage, and strong alcoholic drinks.

The gastric secretion persists to a slight degree in chronic atrophic gastritis and in the severe forms of chronic gastritis where the mucous membrane has undergone structural or atrophic change, though the quantity of hydrochloric acid secreted may be very small. Patients may be allowed meat, very finely divided. An effort should be made to increase the secretory powers of the stomach by prescribing a dietary adapted to this purpose. Meat extractives, condiments, bouillon and extract of beef are recommended. The mode of preparation and of serving the food, if attractive, will have a stimulating action on the gastric functions. The supply of beverages should be limited, especially during the meal, in order to avoid a further dilution of the gastric ferments which are present only in small quantities.

DIET LIST (BOAS).

		Calories.
7 A.M.	200 Gm. milk with 40 Gm. cocoa and 30 Gm. sugar	462 0
	50 Gm. biscuits or zwieback	187 0
10 A.M.	50 Gm. white bread, 30 Gm. butter	343 0
	One egg or 50 Gm. minced ham	100 0
1 P.M.	Soup (30 Gm. tapioca, 10 Gm. butter)	352.6
	One egg, 100 Gm. noodles or spinach, 100 Gm. bean purée, 100 Gm. carrots, 50 Gm. mashed potatoes	282 0
	100 Gm. breast of young chicken, veal cutlet, or veal (steamed), or 100 Gm. squab, game or fish	106 4
	100 Gm. rice omelet, or omelet with ham	288 0
4 P.M.	100 Gm. milk with tea, 20 Gm. sugar	147 5
	25 Gm. biscuits	93.5
8 P.M.	50 Gm. white bread, 30 Gm. butter	343 0
	50 Gm. minced meat	59 5
Total calories		2764.5

Medicinal Treatment.—*Hydrochloric Acid.*—In the treatment of chronic gastritis, medicaments occupy a secondary place compared with diet and hygiene. Of the drug agents, hydrochloric acid is most important and most frequently employed, the object of its use being to supplement the deficiency of the gastric juice (see page 258). In all cases characterized by a diminution or absence of hydrochloric acid, dilute hydrochloric acid should be administered in large doses, 40 to 60 drops, three times a day. The best way to give hydrochloric acid is to add one teaspoonful of the dilute acid to a glassful of water, to be taken three times a day, half an hour after meals— not the whole glass at one time, but in three portions at intervals of one-quarter to one-half hour. Pepsin is frequently given in combination with hydrochloric acid, 0.06 Gm. (1 grain) three times a day; it assists in the process of proteolysis by catalysis, that is, without itself becoming used up or diminished in quantity (see page 258).

Papain and papayotin possess distinct proteolytic properties and are active in neutral, weakly acid or even alkaline solutions. Papayotin peptonizes protein foods. These ferments are indicated in deficient proteolysis with absence of hydrochloric acid, in achylia gastrica, and in gastritis accompanied by subacidity or anacidity. Papayotin and papain are contra-indicated in ulcer and in hyperacidity. The dose is 0.3 to 1 Gm. (5 to 15 grains) after meals.

Pancreatin.—Favorable results from the administration of pancreatin in cases of achylia gastrica, subacid and anacid gastritis, and gastric carcinoma, are often attained. Pancreatin is best administered in doses of 1 to 2 Gm. (15 to 30 grains) in combination with sodium bicarbonate, since it is active only in a neutral or weakly alkaline medium. The preparation should be taken a quarter of an hour after eating. The indication for pancreatin in an anacid stomach consists in the fact that intestinal digestion is thus permitted to begin even before the ingested food passes into the intestine (see page 262).

Stomachics.—We have a number of drugs which possess the property of stimulating the appetite, and others which stimulate the secretory and motor functions of the stomach. Our knowledge of the action of this class of drugs is largely empirical. Loss of appetite is an indication for the administration of stomachics, or bitters, as they are called (see page 266).

The administration of the so-called bitter tonics, gentian, condurango, quassia, and nux vomica, has been found very helpful in chronic gastritis. The fluidextract of condurango, calumba or quassia is to be taken in 20-drop doses three times a day. Tincture of nux vomica may be prescribed in 10-drop doses three times a day, either alone or in combination with the drugs mentioned. These drugs are best given a quarter of an hour before meals, in a little water. Their physiologic action is not well understood. The favorable effect of the so-called bitter tonics or stomachics

is due to their peculiar taste rather than to any direct influence on the gastric mucous membrane. The action of the bitters begins with the sense of taste, before the medicine actually reaches the stomach.

The following is a useful combination of hydrochloric acid with the bitters:

	Gm. or Cc	
R—Tincturae nucis vomicae	12 0	℥ij
Tinctura cinchonae composita	16 0	℥ss
Acidi hydrochlorici diluti	16 0	℥ss
Aquae destillatae q. s. ad	120 0	℥iv

Misce.

Sig. One or two teaspoonfuls in water one-quarter hour before meals.

Orexin is said to possess the property of inducing hunger. It is a stomachic (see page 267), but acts as an irritant to the gastric mucosa; it would therefore be contra-indicated in irritable conditions of the stomach. The tannate of the same base is claimed to be less irritating than the original product. The adult dose is 0.3 to 1 Gm. (5 to 15 grains) in capsule, with a glass of water, one or two hours before meals.

Creosote has also been placed among the stomachics. It causes energetic peristalsis and slightly increases the secretion. It is especially useful in the gastritis of tuberculosis.

	Gm. or Cc	
R—Creosoti	12 0	℥ij
Tincturae gentianae	20 0	℥v
Vini xerici	800 0	Oiss
Alcoholis	200 0	℥vi½

Misce.

Sig. Teaspoonful before meals.

Resorcinol has a stimulating effect on the appetite, as has been demonstrated by clinical experience. It is best taken in solution, either pure or combined with other bitters:

	Gm. or Cc	
R—Fluidextracti condurango	16 0	℥ss
Resorcinolis	4 0	℥j

Misce.

Sig.—Thirty drops four times a day.

Nausea and vomiting may be controlled by administering cerium oxalate, 0.065 to 0.325 Gm. (1 to 5 grains), alone or in combination with bismuth subnitrate or sodium bicarbonate, or by the methods mentioned under Acute Gastritis.

The silver salts are of great value in chronic gastritis (see page 267).

Fermentation may be checked by the use of antiseptic agents, to which a carminative may be added in cases of flatulence. Hydrochloric acid alone may be sufficient; if not, some antiseptic must be employed, such as resorcinol, saccharin, salicylic acid, salicylate of bismuth, menthol, thymol, benzol. These drugs are

best given before meals, either alone or in combination with other medicaments.

	Gm. or Cc.	
R Bismuthi subeylatis	20 0	3v
Resorcinolis	4 0	3j
Acidi acetylsalicylici,		
Salolis	aa 2 0	5ss
Misce et ft. pulv.		
Sig.—One-third teaspoonful three times daily.		
	Gm. or Cc.	
R—Thymolis,		
Resorcinolis	aa 0 75	gr. xij
Misce et ft. caps. no. xx.		
Sig.—One or two capsules before meals.		
	Gm. or Cc.	
R Mentholis	1 0	gr. xv
Alcoholis	20 0	3v
Syrupi	30 0	3j
Misce.		
Sig.—One teaspoonful every hour until relieved.		

Treatment by Gastric Lavage.—Gastric lavage is indicated in cases of chronic gastritis in which there is mucus secretion, disturbance in motility, or fermentation. Mucus should be removed by lavage in the early morning when the stomach is empty. It is well to elevate the irrigator and thus allow the water to enter the stomach with a certain amount of force. In order to avoid over-distention, not more than eight ounces of water should be used at one time. The frequency with which gastric lavage should be practiced must be determined by the amount of mucus in the stomach contents, the adequacy of response to diet and treatment, and the manner in which the patients bear the washing-out process. No fixed schedule can be laid down. Too frequent lavage is liable to do more harm than good. It may be well to give treatments once daily for a week, and thereafter two or three a week, soon lessening the frequency.

Mucus-dissolving drugs may be added to the water after all food particles have been removed. Alkalis which dissolve mucus are: Solution of common salt (1 per cent.); lime-water (5 teaspoonfuls to 1 liter of water); sodium bicarbonate (1 per cent.); Flemer's compound (a mixture of sodium chlorid and sodium carbonate in the proportion of 2 to 1), a heaping teaspoonful to 2 or 3 liters (quarts) of water.

At the termination of lavage distilled water should be employed to clear the stomach, and a weak solution of silver nitrate (0.1 to 0.2 Gm. to 30 Cc.—2 to 3 grains to the ounce) introduced through the tube and allowed to flow out again. Although unpleasant in odor, there is no agent that excels ichthyol (1 per cent.) water as a lavement. Combined with resorcinol it seems to exert a regenerating effect upon epithelia, and is an excellent antiseptic. If fermentation be found in the stomach contents, a weak solution of salicylic acid may be employed as a wash, or a solution (0.1 Gm. to 500 Cc. 2 grains to the pint) of potassium

permanganate. In cases of disturbed gastric motility lavage with lukewarm water should be performed in the evening, before the evening meal, with the patient recumbent. (See Chapter IX.)

Treatment with Mineral Waters.—Waters from the springs of Saratoga, Congress and Kissingen are particularly useful in these gastric affections. They should be taken in small doses on an empty stomach. The artificial waters may be employed when it is not convenient to visit the various resorts.

Good results are often obtained from the use of sodium chlorid waters in subacid chronic gastritis and anacid gastritis with functionally active mucous membrane. The increase in gastric secretion is frequently so marked as to result in a decided improvement and amelioration of symptoms after only a few weeks' treatment.

Mineral water cures are also indicated in those cases of chronic gastritis with normal acidity in which the patients complain of much discomfort, and in which large quantities of mucus are secreted. These cases are best treated by springs similar to Carlsbad.

To avoid the inhibitory action of the Carlsbad waters on gastric secretion, large doses (500 to 600 Cc.) should not be prescribed for a period longer than two weeks, nor smaller doses (200 to 300 Cc.) for more than three or four weeks. Carlsbad water should be taken warm, in the morning, on the fasting stomach, slowly, and in interrupted doses.

Physical Treatment.—Local applications of heat, dry and moist, are often of value in allaying pressure and pain. Compresses, thermophores, or Leiter's stomach application (the moist trunk packing), overnight, are recommended. The Priessnitz bandage is also very valuable (see page 250). The Scotch douche is indicated in the conditions described when they are complicated with atony.

Massage should be adopted in cases not complicated with pyloric stenosis, stagnation, or fermentation. Simple atony is not a contra-indication, but gastric pain is. As described in the chapter on Massage, this manipulation should be performed when the stomach is empty. The purpose of massage is to improve the muscle tonicity and the circulation of the blood. Massage in connection with the use of medicinal agents is useful in some conditions; the drugs are the simple bitters, as in the lavage process.

In chronic gastritis with atony, electric treatment is indicated—the extraventricular faradic current. If there is marked gastralgia, intraventricular galvanization may be employed (see Chapter X).

When chronic gastric catarrh can be definitely traced to congestive conditions related to the portal system, or to diseases of the liver, heart, or lungs, in which the stomach shares in the portal engorgement, treatment of such conditions is especially indicated. When chronic gastritis is secondary to other chronic diseases, these must receive appropriate treatment.

CHAPTER XXIV.

MOTOR INSUFFICIENCY.

ATONY (MYASTHENIA); DILATATION (ISCHOCHYMIA, GASTRECTASIS);
PYLORIC STENOSIS; ACUTE DILATATION OF THE STOMACH.

At one time the opinion prevailed among gastroenterologists that abnormality in size or position of the stomach was largely responsible for motor disturbances. It has been found, however, that greatly dilated and ptotic stomachs do not of necessity cause any disturbance of function. Dilatation of the stomach assumes a pathologic importance only when it interferes with evacuation of the gastric contents into the intestine. The stomach in health should empty itself of a small meal (test breakfast) within an hour and a half, of a large meal (test dinner) in seven hours. The emptying process is, as a rule, accomplished within these limits by either atonic or normal stomachs. In stomachs of both normal and abnormal dimensions the emptying period may be pathologically altered.

Rosenbach introduced the term "motor insufficiency" to designate motor disturbances of the stomach. This term is now in general use. Motor disturbances are classified as motor insufficiency of the first and second degrees.

MOTOR INSUFFICIENCY OF THE FIRST DEGREE (ATONY).

In motor insufficiency of the first degree the evacuation, though complete, is retarded.

Etiology.—Motor insufficiency of the first degree is contingent upon a primary relaxation of the muscular wall of the stomach (myasthenia, atony). Such muscular relaxation may result from irregular modes of living—the frequent overloading of the stomach with food or distending it with fluids; the prolonged use of narcotic drugs (anodynes); or excessive indulgence in tobacco. Idiopathic and hereditary myasthenias have been observed. Motor insufficiency of the first degree may result from acute or chronic diseases, grave anemias, infections, loss of blood, or childbirth. Diseases of the digestive organs, as gastroenteroptosis, chronic gastritis, nervous dyspepsia, chronic intestinal catarrh, chronic constipation, portal congestion, or cholelithiasis, may give rise to primary atony.

Motor insufficiency of the second degree (dilatation) is due to obstruction of the pyloric exit, and is hypertonic rather than atonic.

the gastric walls being hypertrophied from the peristaltic movements of the stomach in its persistent efforts to empty itself. Hypertrophy of the pylorus may result from chronic gastritis, cicatrization of ulcers, slight torsion from gastropptosis, perigastric adhesions and epigastric hernias, hypersecretion with frequent pylorospasm, or repeated injuries in the region of the stomach. The hypertrophic changes in the pylorus in such cases are slowly progressive. These cases, as a rule, pass from mechanical motor insufficiency of the first degree to motor insufficiency of the second degree.

Symptoms.—In motor insufficiency of the first degree (atony) great discomfort may be experienced on the ingestion of food; the pressure symptoms and feeling of fullness may persist for several hours, or in severe cases as long as there is food in the stomach. Patients are apt to be annoyed by eructations, with pyrosis, when hyperacidity is present. The so-called "stomach dizziness" is sometimes experienced in gastric atony complicated with constipation. Patients may complain of many symptoms of neurasthenia, such as fullness in the head, headache, palpitation of the heart, backache, or hypersensitiveness on mental or physical effort. The physician should endeavor to differentiate clearly between motor insufficiency of the first degree (atony) and motor insufficiency of the second degree (dilatation) induced directly by pyloric stenosis.

Diagnosis.—Gastropptosis and atony occur frequently in the same individual. Gastropptosis may sometimes be diagnosticated by inspection when the abdominal walls are thin and relaxed and the stomach is in a condition of peristaltic movement. Permanent and absolute dilatation of the stomach does not occur in primary atony. The atonic muscles may, however, be so greatly distended by the pressure of food as to constitute a condition of transient dilatation of the stomach. Should a person with a normal musculature drink a sufficient quantity of water, the inferior border of the stomach may descend to the level of the umbilicus, as shown by the area of gastric dullness. The atonic stomach, on the other hand, may be so distended by fluids as to throw the lower border below the umbilicus. Splashing sounds elicited when the stomach should be empty go to confirm a diagnosis of atony. The stomach in a condition of atony contains food remnants six or seven hours after the ingestion of a test dinner. It, however, empties itself completely during the night, after a test supper. One hour and a half after a test breakfast the atonic stomach is found to contain food residue (see page 95).

The motility and power of evacuation of the stomach may be demonstrated by Roentgen fluoroscopy. Gastric tonus and atony (Plate XIII, Figs. 3 and 4) can be readily made out by means of the Roentgen ray (see Chapter V).

Examination of the stomach contents withdrawn by means of the stomach tube reveals, in atonic conditions, the presence of free hydrochloric acid in varying quantities, depending upon whether the case is one of simple non-complicated atony or a complication of atony with gastritis or hypersecretion. Should simple atony be protracted for some length of time, the result may be diminished acid secretion. In the absence of gastritis and hypersecretion the acidity usually remains normal for a long time, and the secretion of pepsin and rennin remains normal for a much longer period. Constipation frequently accompanies atony of the stomach.

Treatment. The treatment of motor insufficiency of the first degree should tend to prevent overdistention of the stomach and at the same time improve the muscle tonus. Much may be accomplished by suitable diet, which should be so selected as to make the least demand upon the motor activity of the stomach. The meals should be small in quantity and comparatively frequent. In the atonic as in the normal stomach the liquid portion of the food passes into the duodenum first, then the semisolid, and lastly the solid residues of food. Water leaves the atonic stomach with marked rapidity, so that the amount of water in the tissues of the body is fairly constant. Considering the ease with which the stomach empties itself of liquid and semiliquid foods, these should constitute a large proportion of the diet in atonic states. The stomach can take care of large quantities of liquids so long as they are ingested regularly and in small amounts.

Milk holds first place in the list of foods for the dietetic treatment of gastric atony. In selected cases the milk cure, combined with rest in bed, may be employed for several days. By administering at intervals of two hours 250 to 300 Cc. (3viij x) of milk, 2000 Cc. (2 quarts) may be taken during the twenty-four hours without causing overdistention of the stomach. In addition to milk a variety of preparations with milk may be employed, as cocoa, tea, rice, oatmeal, and corn starch. To prevent fermentation, pure salicylic acid, 0.3 Gm. (5 grains), should be thoroughly mixed with a small quantity of cold milk, the mixture added to the daily quantity of milk (a liter and a half), and the whole boiled. Butter-milk, kefir and peptonized milk are useful adjuncts to the diet.

Diet in Normal Acidity, Hyperacidity and Hypersecretion.—In cases of gastric atony in which the acidity is normal or higher than normal, and in hypersecretion, a strictly protein-fat diet, to obviate the carbohydrate fermentation which would otherwise result from insufficient amylolysis, is to be prescribed. Since protein is quite thoroughly digested in such cases, it is not necessary that it be taken in liquid or semiliquid form; but should gastric ulcer or erosion be suspected, the nutriment must be liquid. It is necessary that the protein food be thoroughly cooked. An extensive

variety of meat and fowl, and dishes prepared from them, as well as jellies, eggs, and soft cheese, may be prescribed.

Fat, owing to its power of diminishing secretion, is indicated in hyperacid conditions. Its use is distinctly advantageous in the treatment of atony. Fat is not classed among the so-called "heavy" foods. Motor insufficiency with increased or normal secretion is benefited, and in some cases a radical cure is accomplished, by a protein-fat diet. All kinds of fat with a low melting-point and pure in quality may be employed. Butter, cream and olive oil are suitable forms of fat; but fat pork and the fat of roast duck or goose should be avoided.

In cases of well-marked atony the physician commences treatment with an exclusive protein-fat diet, and later adds small quantities of carbohydrates so that he has a high protein-fat and low carbohydrate combination. The carbohydrate constituent consists of toast, zwieback, biscuits, rice, leguminous flours prepared in the form of gruels, soups, mashed potatoes—each prepared with as large a quantity of milk and butter as can be used. Green vegetables should be avoided.

Diet in Subacidity and Anacidity.—The principles underlying the dietetic treatment of chronic gastritis apply also in this condition (see Chapter XXIII). The diet should be in all cases liquid or semiliquid. It should contain a large admixture of fat. Meats, if eaten, should be taken in a very finely subdivided condition, and eggs in the form of the light egg dishes. Carbohydrates should be taken in the form of flour soups or leguminous soups and vegetable purées, all of which should be prepared with as much butter and milk as possible. Milk is the best beverage in this class of cases. Alcohol should not be given in gastric atony except in the form of small quantities of mild claret. Coffee should be interdicted, and tea given only in combination with milk. After each meal the patient should rest in the recumbent position, preferably on the right side. If thirst be a troublesome feature of the disease, it may be allayed by the daily administration of two or three enemata of physiologic salt solution of 150 to 200 Cc. (3v-vij) each, thus avoiding distention of the stomach.

Lavage of the Stomach.—Lavage of the stomach is not indicated in atony, inasmuch as the stomach evacuates itself completely, though perhaps tardily. Atony complicated with hypersecretion may be benefited by an occasional lavage. The so-called gastric douche has been recommended in atony, and is said to have the effect of strengthening the muscular coats. When the gastric douche is employed the rinsing may be performed with the aid of Rosenheim's tube (Fig. 21), physiologic salt solution being used in subacidity and Carlsbad salt solution in hyperacidity; the temperature of the liquid may be lowered gradually to 54° F. Should the patient have little or no appetite, the washing process may

be accomplished with an infusion of hops to which has been added a little fluidextract of condurango for its stimulant effect upon the sense of hunger (see page 199).

Medicinal Treatment.—The alkalis are indicated in cases of atony accompanied by hyperacidity or hypersecretion as a complication. The most suitable of these have been found to be magnesium oxid and the double phosphate of ammonium and magnesium. Bicarbonate of sodium has the disadvantage of producing, in combination with the normal acid secretion, too much carbon dioxid, which causes overdistention of the stomach. Atropin is employed for its inhibitory effect in cases of simple non-complicated hyperacidity (see Chapter XX). Hydrochloric acid in combination with pepsin is indicated in subacidity and anacidity. In the presence of fermentation such antifermentative drugs as bismuth, resorcinol, benzoesol, salicylic acid and menthol are to be employed. Strychnin sulphate, 0.001 to 0.006 Gm. ($\frac{1}{80}$ to $\frac{1}{16}$ grain), hypodermically, or extract of nux vomica, 0.008 to 0.05 Gm. ($\frac{1}{4}$ to 1 grain), will increase the muscular tone.

	Gm. or Co.	gr. 1585
R -Extracti nucis vomice	0 1	
Extracti gentiane radicis, q. s.		
Misce et ft pil no xxx.		

Sig -One or two pills three times a day, after meals.

Physical Treatment. Gastric atony has been benefited by the employment of hydrotherapeutic measures. Muscular tonicity has been increased by means of the Scotch douche and cold compresses applied over the gastric region. Massage is indicated in all cases of atony uncomplicated with dilatation or organic stenosis, hyperacidity or hypersecretion; it may be employed even in ptosis of the stomach. The purpose of massage is to improve the muscular tone and aid in the expulsion of the gastric contents into the duodenum. When the purpose is to improve muscular tonicity, massage should be undertaken when the stomach is empty; to aid in emptying the stomach it is, of course, performed when that viscus is filled with food. Electric treatment, consisting of intra- and extraventricular faradization, is also employed as a means of improving the muscular tone. Massage may be employed in conjunction with electric treatment, or electricity and general massage may be employed alternately, to be followed by abdominal massage in cases of arrested intestinal peristalsis (see Chapter X).

Treatment with Mineral Waters.—The use of mineral waters has been found advantageous in the treatment of very mild cases of atony—being selected according to the condition of the gastric secretion; in hyperacidity and in hypersecretion the Saratoga waters and waters from alkaline-carbonate springs should be employed, while, on the other hand, sodium chlorid waters should be used in subacidity and anacidity. The mineral-water

treatment should be employed with great caution, and the waters prescribed in limited quantities in order to avoid overloading the stomach. Mineral waters should not be used in these cases when the patients complain of more or less severe symptoms, but the patients should be sent to the seashore for ocean baths or advised to make climatic changes (see page 252).

In cases of atony in which gastric ulcer, gastritis, ptosis, or neurasthenia is known to be present as a positive factor, the complicating condition should receive treatment as outlined in the respective sections of this work.

MOTOR INSUFFICIENCY OF THE SECOND DEGREE (DILATATION).

Motor insufficiency of the second degree is a chronic condition in which the stomach has lost entirely the ability to expel its contents; that is, food residues remain in the stomach permanently (stagnation); and as a consequence of this chronic condition of gastric insufficiency we have dilatation of the stomach (ischorhymia, gastrectasis). (Plate XIV, Fig. 2.)

Etiology.—The cause of motor insufficiency of the second degree may be either trauma of the muscle fibers of the stomach or pyloric stenosis. It is possible that motor insufficiency of the first degree (atony) may in time be transformed into motor insufficiency of the second degree (dilatation) with stagnation of the stomach contents. Careful clinical and anatomic examinations have shown us that stenosis of the pylorus is the cause of nearly every case of motor insufficiency of the second degree. The lumen of the pylorus may be narrowed from the inside or from the outside; it may be cicatrized and contracted from the healing of gastric ulcers, or there may be cicatricial tissue as a result of healed perforations from biliary calculi. Next to ulcer, carcinoma of the pylorus is the most important and dangerous cause of occlusion of the pyloric lumen. The differentiation is often a very difficult one. A tumor may be so small and smooth as to entirely escape palpation, and there may be nothing but the motor disturbance to indicate stenosis. Polypi and myomata of the pylorus are occasionally met with. Foreign bodies may block the pyloric exit.

Adhesions may change the lumen of the pylorus by displacement and distortion. Cholecystitis may lead to the same result, as it may impede the motility of the pylorus by adhesions, forming the cubwebs of Morris. I recall a case of gastric retention where all the clinical symptoms pointed to a carcinomatous affection. Stagnation was present, with lactic acid, pus and blood in the gastric contents. At operation the stomach was dilated, but there was no tumor. Instead, there was found an infected gall bladder, with gallstones and adhesions. The gallstones were removed, the gall bladder drained, and the patient made a complete recovery.

Spastic stenosis of the pylorus (pylorospasm) is by no means a rare condition; it is caused by the irritating effect of the ingesta upon erosion or fissure of the pylorus, or by an abnormally high degree of gastric acidity (hypersecretion). This kind of closure of the pylorus is at first periodic. When, however, the attacks become more frequent, the effect is permanent stenosis. Spastic closure of the pylorus may also result from hysterical crises. Chronic hyperplasia of the gastric mucous membrane (*état mamelonné*) and hypertrophy of the muscles in the region of the pylorus in chronic gastritis and cirrhosis ventriculi also cause stenosis. Syphilis may become an etiologic factor in chronic hypertrophy of the pylorus.

Adhesions of the stomach to neighboring organs or to abdominal tumors may cause pyloric stenosis by compression or by bending the pylorus upon itself.

A valuable device for ascertaining the patency of the pylorus is the duodenal bucket (Fig. 8). Fastened to a silk string 75 centimeters long, it is swallowed by the patient and allowed to remain overnight. Upon its removal the contents are examined for pancreatic ferment—which, if found, assures us that the bucket has passed through the pylorus and that therefore the pylorus is patent (see page 399). Einhorn has also drawn attention to an important diagnostic point in this connection: if there is an ulcer in the tract covered, the string will be discolored by blood and this will give us a clue to the site of the ulcer (Fig. 84).

Symptoms.—As soon as pyloric stenosis begins to interfere with the free passage of food from the stomach to the duodenum, symptoms of greater or less severity manifest themselves. They may at first be the symptoms of motor insufficiency of the first degree, such as pressure and a sense of fulness after eating; and the desire for food is easily satiated. Eventually the pressure symptoms become aggravated in proportion to the increasing stenosis of the pylorus, the stomach becomes distended, and pain is caused by the incessant attempt of the gastric muscles to overcome the obstruction to the pyloric exit. When the obstruction becomes so pronounced as to effect a closure of the pylorus, the food remains in the stomach and stagnation results. By far the most important symptom of gastric retention is vomiting, which is usually profuse. At first it does not occur often, but the intervals continue to grow shorter until at last large quantities, apparently larger than those ingested, are vomited every day. The vomitus will contain food remnants many days old, for food that is not readily digestible may remain in the stomach for days.

On standing in a sedimentation glass, the vomitus usually separates into three layers. The solid particles, being the heaviest, sink to the bottom; the fluid above is cloudy, and the top layer consists of more or less viscid mucus permeated by gas bubbles.

This stratification in three layers is thoroughly characteristic of all forms of gastric retention which are due to or associated with stenosis of the pylorus.

The vomiting of malignant stenosis is totally different, especially after the affection has reached an advanced stage. The vomitus is no longer dilute, but viscid—like a thick soup—and permeated by mucous masses, everything being so closely intermixed that it is difficult to diffuse the mass with water. The odor is peculiarly mouldy, sometimes absolutely putrid, like decomposed tissue. The food remnants are almost unchanged. Meat can be found days after being taken into the stomach, and even farinaceous food is undigested. The appearance of the gastric contents is so characteristic as to be almost sufficient of itself to determine the diagnosis.

The appetite, fair at first, diminishes with the increasing stagnation. Patients in the meantime complain of severe thirst. The body becomes impoverished for fluid, since the stomach cannot absorb water. This condition is indicated by the remarkably small quantities of urine excreted and by hard impacted fecal matter. The pyloric stenosis is accompanied by pronounced emaciation. The skin is dry and drawn because of the small amount of water in the tissues. Patients, as a rule, complain of dizziness, lassitude, inability to work, and somnolence. When the decomposed gastric contents pass into the intestine, pronounced gaseous fermentation arises, producing distention of the bowel, with abdominal pains and headache. Gastrogenic diarrhea may be brought on by the decomposed gastric contents irritating the bowel (see Chapter XXXVIII).

Diagnosis.—Dilatation of the stomach from stenosis of the pylorus may assume marked dimensions. It is a matter of diagnostic importance to ascertain the degree of dilatation. A dilated stomach has a characteristic form which is easily demonstrated by the Roentgen ray (see Chapter V). In obstruction of the pylorus the shadow near the stenosis is round and blunt (see Plate XVI, Fig. 1). Apart from the anamnesis, the diagnosis is facilitated by the presence of abnormal peristaltic movements (visible contractions) of the stomach, by the signs of motor insufficiency of the second degree, and by examination of the stomach contents. Motor insufficiency is indicated by the nature of the food remnants in the stomach in the morning after a night's fast. Fermentation is always present, its extent depending upon the degree of stenosis of the pylorus. In benign stenosis the stomach contents are acid, owing to the presence of hydrochloric and organic acids, such as acetic and butyric, the latter resulting from fermentation; sulphuretted hydrogen gas also is present, arising from the decomposition of protein matter. In malignant stenosis (carcinoma), lactic acid predominates, but hydrochloric acid may also be present

for a considerable time, especially at the beginning of the carcinomatous process. The finding of sarcinae is of diagnostic significance in benign stenosis of the pylorus; the presence of lactic acid bacilli will aid in the confirmation of malignant stenosis. When the stagnating gastric contents become strongly acid, the urine may be found to be alkaline in reaction, with a resultant lowering of the percentage of chlorids in the body. The presence of bile in the gastric contents favors a diagnosis of stenosis of the duodenum. Gastric hemorrhage may occur in either malignant or benign stenosis of the pylorus. The stomach contents and the test-diet stool are characteristic (see page 95 and Chapter IV).

Treatment.—The treatment of this condition is essentially dietetic. The diet should be such as to make the least possible demand upon the motor activity of the stomach. It should not be larger in amount than is absolutely essential, and it should be ingested in a form most easy of expulsion from the stomach into the duodenum. The diet in this class of cases resembles that advised in atony. The condition of the secretory function must be carefully estimated. Fat may be prescribed along with protein when the secretion of hydrochloric acid is either normal or above normal. Carbohydrates I prescribe in as small amounts as can be got along with, and give them in the most soluble form possible, preferably dextrinized. The artificial protein preparations are indicated in this condition. Green vegetables should be avoided, even in the form of purées. The food should be liquid or semisolid in consistency.

Beverages should be restricted to the lowest practicable limit, and should consist of drinks with a nutritive value, such as milk or cocoa. If the patient can tolerate it, the oil cure recommended by Cohnheim may be employed with advantage (see page 273). This consists in the patient drinking, or in having introduced by means of the stomach tube, three times a day, before meals, 50 to 60 Cc. (Siss-ij) of pure olive oil, at body temperature. If lavage is a part of the general treatment, 100 to 200 Cc. (Sijj-vij) of oil may be introduced at the conclusion of each lavage, when the stomach will be sure to be empty. The oil has an antispasmodic action and serves as a coating, being especially useful if fissures, erosions or ulcers are present. Oil has the additional advantage of diminishing the secretion in cases of hypersecretion and hyperacidity. The oil treatment is recommended particularly in spastic contraction of the pylorus.

To Allay the Thirst.—An attempt should be made to allay thirst, which is often very distressing, by moistening the lips and the cavity of the mouth. The mouth should be frequently rinsed with cold aromatic waters. Small pieces of ice may be given, but the water should not be swallowed. When this method of allaying the thirst fails, recourse must be had to rectal enemata. Water

is readily absorbed by the rectum and colon, especially when the body has become much impoverished for want of fluids. Eight to ten ounces of lukewarm water should be allowed to flow into the rectum through a soft-rubber tube, preferably by the drop method (see page 239), so that the patient may retain as much of the fluid as possible until absorption takes place. Normal salt solution may be used instead of pure water.

Rectal Alimentation. When food cannot be retained in the stomach, rectal alimentation may be employed with advantage and continued exclusively for eight to fourteen days (see page 243). The physiologic rest of the stomach afforded by this method of feeding is usually followed by marked improvement in the gastric symptoms; the improvement is often so pronounced as to permit of a resumption of feeding by mouth. Feeding by mouth should, however, be resumed very gradually, and as the power of gastric digestion increases the number and quantity of nutrient enemata may be as gradually decreased.

Subcutaneous Nutrition.—When rectal alimentation fails, subcutaneous nutrition remains as a last resort. It has been shown that grape-sugar solutions are well borne when administered hypodermically; the injection is, however, accompanied by much pain. About 100 Cc. (3ij) of a 10-per-cent. solution of grape-sugar may be introduced by means of a cannula connected with a funnel. Injections of oil, such as olive oil or oil of sesame, are said to be less painful. Oil may be injected in quantities up to 100 Cc. by means of a funnel and cannula or the syringe. Nutrition by hypodermic injection is only to be thought of when no other method of feeding is practicable. Subcutaneous injection of water (hypodermoclysis) in cases in which the quantity of water in the tissues has become greatly reduced has been found very efficacious; the water is usually given as normal salt solution; from 1 to 1½ liters (2 to 3 pints) may be administered, and the injection repeated.

Treatment by Lavage.—Routine washing of the stomach is indicated in all cases of motor insufficiency in which that viscus does not completely empty itself of its contents during the night's fast. The stomach should be emptied and relieved of the retained food remnants. The most satisfactory results are obtained by the use of gastric lavage in the rare forms of atonic stagnation with insufficiency, and in spastic stenosis of the pylorus. After a continued course of gastric lavage the dilated stomach has been found to approximate the normal, and the gastric muscles have shown marked improvement in tone; especially is this the case in benign stenosis of the pylorus. We do not get this improvement in cases of malignant stenosis. It is sometimes possible, however, by means of lavage, to improve the *pyloritis* and thus arrest the progress of pyloric stenosis. A proper time for the performance of

lavage is in the evening, before supper, so that the stomach may be relieved of undigested and decomposed food remnants before another meal is taken.

Gastric lavage is usually followed by a marked amelioration of the subjective symptoms. The appetite increases, pain ceases, vomiting disappears, and thirst is diminished, while at the same time the urinary secretion becomes normal in amount. All this improvement should take place within three or four weeks, otherwise the prognosis is not good.

Mechanical Treatment.—When dilatation and ptosis exist, improvement sometimes follows the application of properly fitting abdominal bandages which assure support to the stomach. The reader is referred to the chapter on Gastroenteroptosis for details in regard to this mechanical treatment.

Physical Treatment.—The galvanic current is indicated in those rare forms of motor insufficiency of the second degree in which there is no obstruction at the pylorus. In pyloric stenosis the peristaltic movements of the stomach are accelerated, thus rendering unnecessary any extraneous aid for the purpose of improving muscular tone. Massage of the stomach may be practiced in the treatment of atonic varieties of motor insufficiency of the second degree. It should not be employed when the stomach contains food remnants, but only after the decomposing material has been removed by lavage.

Mineral Waters.—The mineral-water cures, so called, are contraindicated in motor insufficiency of the second degree in the presence of stagnation, inasmuch as their employment would only serve to increase the amount of fluid in the overburdened stomach.

Medicinal Treatment.—The internal administration of drugs is probably the least important factor in the treatment of motor insufficiency with stagnation, since the drugs must come in contact with decomposed food remnants in the stomach. Strychnin sulphate may be administered hypodermically. As an antifermentative in cases of gastric distention, either sodium salicylate or magnesium salicylate, 1 to 3 Gm. (15 to 45 grains) in divided doses for the twenty-four hours, is useful. One Cc. (15 minims) of dilute hydrochloric acid may be administered several times a day for an extended period, in order to counteract fermentation caused by the presence of lactic and butyric acids. The vegetable bitters, such as condurango and quassia, are sometimes useful. In spastic contraction of the pylorus brought on by hyperacidity and hypersecretion, alkalis, astringents or atropin sulphate may be administered as indicated in hyperacid conditions of secretion. The latter drug should be administered immediately after gastric lavage, in order that it may come in contact with the empty stomach, thus ensuring absorption.

When pyloric stenosis can be traced to a syphilitic cause, specific

treatment will be productive of good results. In cases where the stenosis is due to gastric carcinoma, little can be hoped for from a course of internal medication.

Some writers have reported favorable results from the administration of thiosinamin. Thiosinamin has been superseded by the discovery, by Mendel, of an analogous preparation, fibrolysin (thiosinamin and sodium salicylate). Fibrolysin is supplied, in brown glass bulbs, sterile and ready for use; each bulb or ampoule contains 2.3 Cc. of a solution of $1\frac{1}{2}$ parts fibrolysin to $8\frac{1}{2}$ parts distilled water, corresponding to 0.2 Gm. thiosinamin. Fibrolysin should be injected in the intrascapular region directly into the muscles. The effect is similar to that of thiosinamin, namely, the softening and rendering elastic of cicatricial tissue, thereby preventing the contraction which results from cicatrix formation. I would advise that a trial be made of these agents, especially in comparatively vigorous patients who are able to take and retain nourishment by mouth. In severe cases, however, in which stenosis is well marked and associated with emesis and pronounced emaciation, such treatment will not be successful; to adopt it would simply be temporizing instead of giving the patient the benefit of early surgical intervention.

Treatment of Stenosis of the Pylorus.—We should direct our treatment likewise to pyloric stenosis. Success is often attained by combating the causes of pylorospasm, which usually consist of hyperacidity, hypersecretion, or fissures and ulcers in the region of the pyloric exit. This treatment may be dietetic or medicinal, or it may consist of lavage or the oil cure already described (see page 481). Organic stenosis yields with much greater difficulty, if at all, to internal medication.

Einhorn has constructed a special pyloric dilator (Fig. 82) which is useful in stenosis of the pylorus and in spasmodic contraction of the pylorus. This mode of treatment will probably find application in specially selected cases, principally of pylorospasm due to gastric ulcer or to remote reflexes (see page 400). Benign pyloric strictures can also be widened with the pyloric dilator. The results from this method of treatment are very encouraging. Internal stretching of the pylorus should be attempted in all benign cases before considering surgical intervention. Its use is imperative in benign pyloric strictures complicated with affections of the heart or kidneys or grave lesions of the liver; for in these cases the mortality of operative measures is so great as almost to forbid it, while the widening of the pylorus by the internal route can be done practically without danger. A modification of the pyloric dilator has for its object stretching of the pylorus *in situ*, which is impossible with the other instruments. This modified dilator is provided with a double-canal tube and two balloons, which can be inflated separately. When this tube

is in the duodenum the end balloon is inflated and pulled up to the pylorus, then allowed to recede about half an inch. Thereupon the second balloon, collapsed, and lying just within the pylorus, is inflated and left in position for about one minute. This should be practiced about once a week. The technic is given in detail on page 400.

I have used the pyloric dilator with remarkable success in one case of benign stenosis of the pylorus. The patient was a man forty-eight years of age. He had been sick nine years. For the last five years the great discomfort he experienced compelled him to wash out his stomach frequently. Pain, and sometimes vomiting, would come on about two hours after meals. At times he would vomit enormous quantities of food that he had taken days before. He had lost thirty pounds in weight; was tired quickly; had headache and was constipated. His stomach contents showed stagnation with hyperchlorhydria. No Oppler-Boas bacilli could be found, but there was an abundance of sarcinae. The feces contained no occult blood. The use of the pyloric dilator brought about an apparently complete recovery. The dilator was used only three times during twelve days. The patient improved so rapidly that it was unnecessary to do any more stretching of the pylorus. He regained his weight and strength. It is now over two years since the last stretching.

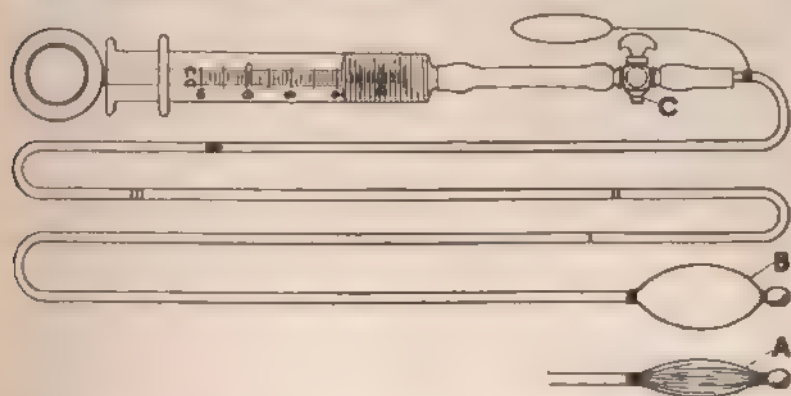


FIG. 82 Einhorn pyloric dilator. A, rubber bag with gauze envelope, collapsed. B, rubber bag with gauze envelope inflated with air; C, stopcock.

Surgical Treatment. Should internal medication combined with the treatment outlined fail after a reasonable time, the patient should be referred to the surgeon. Gastroenterostomy is usually followed by favorable and permanent results in benign stenosis of the pylorus. For the treatment of carcinoma see Chapter XXIX.

Gastric Tetany.—Kussmaul was the first to draw our attention to the fact that in certain cases of dilatation of the stomach tetanus

noid spasms occur. We now know that there are several conditions of the gastro-intestinal tract which may cause convulsive attacks. Robson and Moynihan believe that the appropriate treatment in all cases of gastric tetany is surgical. They conclude that in almost all cases there is a mechanical obstruction to the onward passage of food. It is this stenosis which causes dilatation and hypertrophy of the stomach. To relieve the obstruction and to prevent stagnation of the stomach contents, surgical measures are necessary.

ACUTE DILATATION OF THE STOMACH.

This condition is noted especially after laparotomies, injuries, chloroform narcosis, torsion of the duodenum or of the mesentery, dietetic errors, severe infectious diseases such as pneumonia and scarlet fever, and chronic exhausting diseases interfering with the internal secretions. It is assumed by some that dilatation of the stomach follows any condition that causes acidosis of the stomach wall. The main feature is the immense distention of the stomach and upper part of the duodenum. The dilated portion is outlined abruptly where the mesenteric artery crosses and compresses the duodenum. The distended stomach exerts traction on the mesentery below the duodenum, thus tightening the pressure on the latter and starting a dangerous vicious circle. The swallowing of air after operation progressively distends the stomach and frequently causes a condition of dilatation. This condition is evidently the first phase of an "arteriomesenteric occlusion of the duodenum" (see page 743). Aerophagy is occasionally the primary factor in some of these surgical and medical cases.

When acute dilatation of the stomach takes place in a previously healthy person the clinical symptoms are, as a rule, most pronounced. These symptoms consist of vomiting, intermittent pains, collapse, feeble pulse, accelerated respiration, and constipation. When the condition does not readily clear up, it must always be regarded as very grave.

Intestinal obstruction invariably causes, as one of its early symptoms, dilatation of the stomach (see page 745).

Treatment. When a diagnosis of acute dilatation has been made, the stomach should be emptied and lavage performed as frequently as indicated. No food should be given by mouth; the nourishment should consist of nutritive enemata. Otherwise treatment should be directed to the reestablishment of normal gastric peristalsis. Rectal enemata (proctocolysis) consisting of large quantities of physiologic salt solution are indicated. In selected cases strychnin sulphate and atropin should be employed. Collapse should be treated by means of stimulants and normal salt solution.

Early, prolonged and repeated lavage reestablishes the normal mechanism of digestion. It relieves the stomach and duodenum of gas, stagnated fluid, and the residues of secretion. The removal of this toxic material is conducive to recovery, since the stomach then contracts, thus relieving the arteriomesenteric pressure. It may be necessary to continue this process for many hours. Cathartics are absolutely contra-indicated. When the trouble is due to abnormal fermentations or overloading, the washing-out of the stomach has a direct curative action, and even when the trouble is of other origin it has a very useful palliative effect. The patient may be placed in the Trendelenburg position, a stomach tube being used for drainage. Epinephrin is especially indicated when there is reason to assume that the severe symptoms are the result of acute exhaustion of adrenal function. Patients should always lie on the right side to assist in emptying the stomach contents through the pylorus. When these measures fail, gastroenterostomy is indicated.

CHAPTER XXV.

GASTRIC ULCER.

ULCUS VENTRICULI—ROUND ULCER—PEPTIC ULCER—PERFORATING GASTRIC ULCER.

GASTRIC ulcer is a localized lesion of the mucous membrane of the stomach. It is characterized by a sharp, well-defined outline, more or less deep destruction of the mucosa, and by no tendency to heal. The lesion gives rise to one or more characteristic symptoms—pain, vomiting, hematemesis. Gastric ulcer was first described by Cruveilhier in 1829.

Pathology.—Gastric ulcer is usually round or oval in shape. In some instances several ulcers may become confluent and thus form a larger one with an irregular border. Owing to the tendency of the ulcer, which is at first superficial (florid ulcer), to penetrate deeply, the base is frequently the muscular or serous coat of the stomach. In "perforating ulcer" the base is one of the adjacent viscera, bound to the stomach by adhesions. The ulcer is funnel-shaped, with the base as the apex. As a rule ulcers do not attain a size much larger than a dime, though some of the confluent variety have measured ten centimeters (over three inches) in their greatest diameter. An ulcer the size of a pea may exhibit all the characteristic symptoms of this pathologic condition. The typical gastric ulcer has a punched-out appearance.

Chronic ulcer consists of excavations in the gastric wall having either the mucosa, musculature, serosa or perigastric structures as their base. The mucosa may recede or overhang the base, which consists of scar tissue, radiating from the center. All chronic ulcers are protected with a callus which forms the base of the ulcer. In carcinomatous degeneration the carcinomatous cells are usually found in the overhanging mucosa.

On microscopic section of recent ulcers the margins show the ducts of the gastric glands cut off toward the base of the ulcer. In chronic ulcers, owing to a reactive inflammation at the periphery, a thickening of connective tissue is formed there (callous ulcer) which may be palpated—especially if the ulcer is located near the pylorus. Apart from the inflammation surrounding the edges of the ulcer, the remainder of the gastric mucosa is likely to be normal.

In the acute form of the lesion the necrotic process may be so

rapid that the thin serous coat is perforated (perforating ulcer), or a vessel may be so eroded as to occasion severe hemorrhage, with a fatal termination.

Etiology.—The lesser curvature seems to be the favorite seat of ulceration. In about 86 per cent. of cases the ulcer is situated on the posterior surface of the lesser curvature and at the pyloric sac—parts of the stomach which together form a segment of less than half the total surface of the viscus. This portion of the stomach is subjected to the greatest irritation from the moving mass of gastric contents before the latter are entirely reduced to liquid form. Another explanation is that these parts of the gastric mucosa may be insufficiently nourished, in consequence of disturbances of circulation due to spastic contraction (vagotonia, see page 388), so that they are attacked by the digestive activity of the normal gastric juice, and the so-called peptic ulcer is the result. Such disturbances in circulation may also be caused by severe trauma, simple injury to the stomach, or traumatic influences extending over a prolonged period, such as pressure from corsets, the wearing of belts by workmen, continuous work in a bent position, or the tasting of superheated dishes by cooks. Insufficient nourishment, induced by circulatory disturbances, is also traceable to embolism or thrombosis of the small arteries supplying the lesser curvature of the stomach; specific endarteritis; venous stagnation, from chronic inflammatory processes of the mucous membrane; and altered composition of the blood (anemia, chlorosis). A focus of infection (oral sepsis) may be the predominating factor in the causation of gastric ulcer (see Duodenal Ulcer, Chapter XLI). From typical indurated ulcers of the stomach, streptococci have been isolated, suggesting a bacterial origin for the ulceration; and these same streptococci (*viridans*) injected intravenously in animals have caused ulcerations of the stomach. Autodigestion never occurs in the gastro-intestinal tract below the field of action of the peptic ferment. There are no "tryptic ulcers," like the "peptic ulcers." This is explained by the ability of the peptic ferment to digest raw connective tissue, which the tryptic ferment is unable to do (see page 119). The initial lesion of peptic ulcer may also be due to a derangement of the internal secretions (see page 388).

Frequency.—Lebert found one case of gastric ulcer in 200 autopsies. Grunfeld places the number at 20 per cent. These are the extremes. Brinton found 5 cases in 100 autopsies; Berthold, one in every 37, or in that proportion. In a clinical study of 1000 cases of gastric disturbances of various kinds, Friedenwald found that 7.8 per cent. were affected with ulcer of the stomach or duodenum.

Sex Predisposition and Age.—Gastric and duodenal ulcers occur much more frequently in males than in females. Of chronic

gastric ulcer coming to operation, Mayo reports 29 per cent. females and 71 per cent. males. These ulcers have been observed at an early age, Lees having found perforation of the stomach in children aged eight and nine years. Habershon, in an analysis of 201 cases, noted the earliest age at which gastric ulcer occurred to be ten years (the patient a girl); several children (girls) suffered from gastric hemorrhage at fourteen, others at fifteen and sixteen; the oldest patient was a man aged seventy-one. This writer found the disease to be most frequent in the period between twenty and fifty years. In women the period of liability was noted to begin earlier than in men, and to reach its maximum at twenty-five to thirty. In men the earliest case occurred at the age of twenty.

Effect of Healing.—Deep ulcer heals by cicatrization. The scar is pale and star-shaped, with a puckering of the surrounding mucous membrane. Cicatrization and scarring may eventually lead to deformity of the stomach, producing the so-called hour-glass contraction. Hour-glass stomach is readily diagnosed by means of the Roentgen ray (see Plate XV, Fig. 2). More often, however, there is interference with gastric movement and function by adhesions to neighboring organs. Stenosis of the pylorus, with resultant obstruction and dilatation of the stomach, occurs in the healing of ulcers near the pyloric exit. The involvement of the pneumogastric nerve in the scar occasionally gives rise to intense suffering.

Symptoms.—The symptoms of gastric ulcer are at first ill-defined, resembling those of gastritis; much will depend upon the size, shape, depth and location of the ulcer. There is more or less discomfort after partaking of food; and later on in the course of the disease nausea may develop, or more often regurgitation of food, or vomiting. A periodic boring pain is characteristic of well-established gastric ulcer; it comes on always within an hour after eating, sometimes as soon as the food is ingested, and may be aggravated by the character of the latter, especially when not well masticated. Fruits and vegetables favor the development of the gastric pain, whereas proteins may relieve it. (The pain in duodenal ulcer appears at any time from two to three hours after eating, and is relieved by the taking of food.) Liquid food is borne much better than solid. Pain in these conditions varies in intensity from the slightest pressure discomfort to paroxysmal agony. The painful seizures are particularly frequent and severe in gastric ulcer complicated with hypersecretion, hyperacidity, and pylorospasm (see page 398). The appetite is usually good, but, since eating is followed by such distressing symptoms, patients are inclined to eat as little as possible and consequently they become much emaciated as the condition progresses.

It is now established that the sensation of hunger is induced by a type of tonic and peristaltic contractions of the empty or

nearly empty stomach. These gastric hunger contractions occur with a rhythm and intensity that have no relation to the peculiar character of the food ingested. The tension of excessive contractions on sensory nerves rendered hyperexcitable by the presence of gastric ulcer will result in more or less severe pain; and there is evidence that the pains of gastric and duodenal ulcers are contraction pains arising in the stomach, pylorus, or duodenum. They have been correctly named hunger pains. These pains are temporarily relieved by any measure which inhibits or decreases gastric tonus. This result can be brought about by the ingestion of food, alkalis, or water.

Localization of Pain.—The location of the pain corresponds, as a rule, to the center of the epigastrium—at the median line, just below the ensiform cartilage. The portion of the epigastric region to which the pain is referred forms a circular area of less than two inches in diameter. The pain is increased on pressure.

Cruveilhier first described the dorsal pain, which appears a few weeks or months later than the epigastric pain. This pain, which is of a gnawing character, is to the left of the spine and at about the eighth or ninth dorsal vertebra. It may extend occasionally to the first or second lumbar vertebra. Boas has drawn attention to a dorsal point of pressure at the level of the tenth to the twelfth dorsal vertebra, with a lateral extension of two or three centimeters and a height of one to four centimeters. This pressure point is usually left of the median line.

Vomiting.—Vomiting usually occurs an hour or two after meals, or when the pain is at its height; and the pain is, as a rule, relieved by the emesis. The vomitus consists of either gastric juice or watery fluid containing partially digested food remnants. Instead of vomiting, the patient may have attacks of nausea.

Hemorrhage.—Hemorrhage, if slight, may pass unnoticed; but if there is any considerable quantity of blood in the vomitus it will impart to the latter a red or coffee-brown appearance. When it is not possible to detect the presence of blood from the macroscopic appearance of the vomitus or dejecta, it is well, in suspicious cases, to resort to Weber's test (see page 86) for occult blood. The examination of both gastric contents and feces by means of this test to detect concealed hemorrhages is of great assistance. The benzidin test and, more recently, the phenolphthalein test have been devised, which give more characteristic reactions (see pages 123 and 124).

When gastric hemorrhage is profuse, the patient will experience a feeling of giddiness, weakness, syncope, and extreme thirst. Among the objective symptoms is pallor, the degree of which will depend upon the amount of blood lost. If the effusion of blood in the stomach is considerable, hematemesis or melena may occur. Hematemesis as a symptom is not necessary to the diagnosis of

gastric ulcer, though it aids in confirming the diagnosis. It occurs in about half the cases.

Perforation.—Perforation is one of the most frequent causes of death from gastric ulcer. The extravasation of gastric contents into the peritoneal cavity is attended by sudden and severe abdominal pain similar to that brought on by exertion or by some dietetic error. Syncope and collapse, weak, running pulse, and peritonitis with a fatal termination, is the usual result of perforation of a full stomach. Disappearance of hepatic dulness is of notable diagnostic value. An early sign of perforation of the stomach is tenderness of the pouch of Douglas. The stomach contents frequently flow down and accumulate here, which explains the extreme tenderness at this point. (If the stomach be empty, the symptoms of perforation are comparatively unimportant.) In the event of extravasation of gastric contents into the peritoneal cavity, life is saved only by prompt resort to operative intervention. In operations performed within ten hours after perforation the mortality is 28 per cent.; if the operation be delayed for more than twenty-four hours the mortality rises to 65 per cent.; after thirty-six hours, to 87 per cent. Later, operation is practically hopeless. (See Plate XIV, Figs. 3 and 4, and Plate XV, Fig. 1.)

In perforations in which diffuse infection does not take place, owing to the fact that there was no food in the stomach, adhesions are formed with neighboring viscera. This subject is discussed under the heading Perigastritis, Chapter XXVII.

Willan draws attention to the presence of a ring of constriction across the abdomen at the level of the lower margins of the ribs as a helpful diagnostic sign in cases of perforated gastric ulcer. The appearance is as if the abdomen were constricted above the level of the umbilicus and below the transpyloric plane by an invisible rope, which represents the level of the lowermost limits of the costal arch and may be termed the "infrascostal" line. The constriction does not disappear with general anesthesia, but does disappear when that condition has advanced to the stage of general abdominal distention. There is no marked hyperesthesia, and the patient has no feeling of tightness at the site of the constriction. It is assumed that the powerful impulses resulting from the perforation are conveyed by the sympathetic fibers to the celiac plexus and from there to the spinal cord, whence the whole nervous system is probably involved.

Appetite. The appetite is apparently not affected by the presence of gastric ulcer, though patients are apt to eat but sparingly through fear of the pain which the act induces. Patients complain of constant hunger, owing to this inability to satisfy the appetite on account of the excessive quantity of hydrochloric acid present. This acidity is best estimated by an analysis of the stomach contents (see page 67).

Complications and Sequelae.—Manges, from the viewpoint of origin, classifies the complications and sequelae of gastric ulcer as (1) intragastric; (2) extragastric. Among the intragastric are: (a) hemorrhage; (b) profound anemia; (c) interference with motility of the stomach (if the lesion extends deeply into the muscularis); (d) stenosis of the cardia, pylorus, body of the stomach (hour-glass contraction); (e) gastrorrhea, with its various complications, such as tetany; (f) carcinoma. The extragastric complications include: (a) perforation, free and with adhesions, possibly suppuration, also subphrenic and other abscesses, fistulae of various kinds; (b) general emphysema; (c) perigastritis, with localized thickening of the serosa, adhesions to various organs, displacement or distortion of the stomach. These sequelae are dealt with in this and other chapters.

Diagnosis.—A probable diagnosis of gastric ulcer may be made from the fact of profuse hematemesis, if carcinoma of the stomach and obstruction to the portal circulation can be excluded. In every case, unless the ulcer is cicatrized, occult blood is demonstrable in the feces. Pain appearing shortly after eating and lasting for two or three hours is of diagnostic import, especially if there is a circumscribed spot in the epigastric region that is painful to pressure, or a similar sensitive area to the left of the eighth or ninth dorsal vertebra. Vomiting occurring shortly after meals in patients who have recently become pale and anemic will justify a probable diagnosis of gastric ulcer. Should the vomiting culminate in hematemesis or melena, and cause a cessation of pain, the physician may reasonably conclude that the lesion is gastric ulcer. The relief of pain afforded by orthoform is of diagnostic value (see page 270).

The development of chronic gastric ulcer is frequently due to vagotonia, or increased irritability of the vagus (see page 388).

For determining the presence and location of gastric or duodenal ulcer, the "string test" devised by Einhorn has been found valuable (Figs. 83 and 84). The stomach being empty, the patient swallows, preferably at night, the Einhorn duodenal bucket attached to a braided silk string 85 centimeters long, which is to be knotted just before removal at the level of the upper incisor teeth. A loop at the upper end of the string is placed over the ear to prevent the upper part of the string from passing into the stomach. The bucket is withdrawn on the following morning and the string examined for a red or brown stain. The lower end of it is found to be yellow or greenish-yellow, and the bucket contains bile mixed with mucus, provided it has passed the pylorus—which it invariably does in from two to eight hours if there is no obstruction at the pylorus. Should the bucket fail to pass into the duodenum, a smaller one is used the succeeding night, and in this manner an approximate idea of the caliber of the pylorus may be gained.

By measuring the distance from the knot in the string to the red or brown stain (should there be one), we are able to definitely localize the ulcer. If the stain is 39 to 42 centimeters from the incisor teeth, the ulcer is located at the cardia; if 44 to 54 centimeters, at the lesser curvature; if 55 to 56 centimeters, at the pylorus; and if over 57 centimeters, in the duodenum. I have substituted a large porcelain bead for the Einhorn bucket and find that it serves equally well. If this test be made several times on one individual, and each time a red or brown stain is found at about the same distance from the teeth, the clinician may be sure that a localized lesion of the gastric mucosa exists, which is probably ulcer. This test gains in value the more I use it.

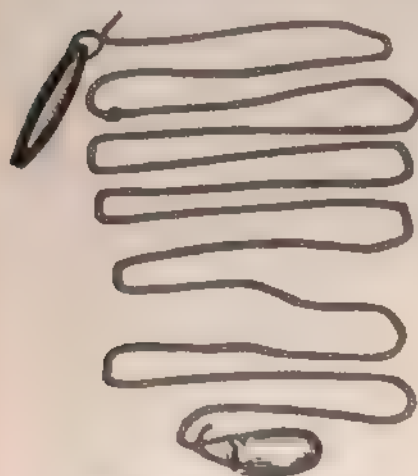


FIG. 83.—A negative string.

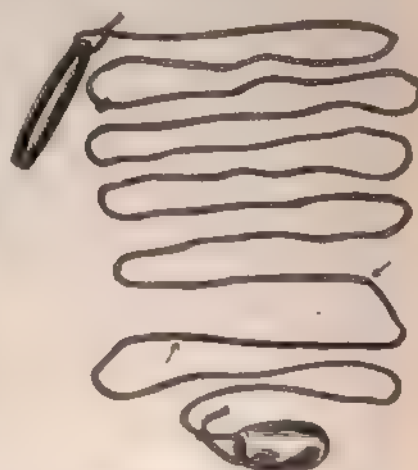


FIG. 84.—A positive string.

Roentgenography is of great assistance in the diagnosis and location of chronic gastric ulcer. In the superficial or florid cases it does not help as much. In all cases of the callous type roentgenologic diagnosis usually proves correct, as shown by operation (see page 141).

Prognosis. With a better understanding of the etiology and pathology of gastric ulcer, as well as improved methods of treatment, the outlook for complete recovery is much more favorable than formerly. The reason there are not more successes in the treatment of this pathologic condition is that patients frequently present such indefinite symptoms that the nature of the disease is obscured and improper treatment instituted. The further fact that physicians too often fail to insist upon the discipline necessary for the accomplishment of the best results, but content themselves with prescribing a few dietary rules and some harmless drug, has resulted in a chronicity that at times resists rational

treatment. The physician should insist very strongly on the rest cure. The older the ulcer the more unfavorable the prognosis. Peptic ulcer having its base on the serous membrane or on some organ in close proximity to the stomach will resist all medical treatment; surgery is the only recourse in such cases (callous ulcer). The location of the ulcer is a matter of importance: in ulcers of the pylorus, owing to the fact that they tend to produce cicatricial stenosis, sometimes the only hope of recovery lies in operative treatment. When the ulcers are deep we are apt to have such complications as hemorrhage from perforation, adhesions to the spleen if the ulcer happens to be located in the fundus, and perigastritis. With hypersecretion as an accompaniment the prognosis of gastric ulcer is less favorable than when there is simple hyperacidity (see page 97).

Treatment.—Prophylaxis.—Hygiene of the mouth is most important (see page 296). A properly selected diet will do much to prevent the occurrence of ulcer of the stomach. An absolute milk diet should be prescribed as soon as the first symptoms of the disease become manifest. Care should be exercised to avoid extremes of temperature in food. An effort should be made to overcome the hyperchlorhydria which is an important etiologic factor in gastric ulcer. The anemia which is a frequent accompaniment of the disease should likewise be treated.

Leube-Ziemssen Treatment.—A therapeutic procedure suitable to slight or moderately severe cases of gastric ulcer uncomplicated by hemorrhage is the Leube-Ziemssen treatment. After the diagnosis has been confirmed, the patient is given, for the first fourteen days, complete rest in bed. Every morning, an hour before partaking of food, he is given one-quarter liter ($\frac{1}{2}$ pint) of Carlsbad Muhlbrunnen water (at 90° F.) in which is dissolved 5 to 10 Gm. (75 to 150 grains) of natural or artificial Carlsbad salts. It is also advisable to dissolve 10 grams of Carlsbad salts in a quarter of a liter of pure water at a temperature of 90° F., to be sipped at intervals.

Hot fomentations are applied over the epigastrium during the day. For this purpose mashed potato poultices or linseed poultices are good. A piece of clean flannel cloth should be interposed between the skin and the poultice. In using thermophores or electric heating pads, which furnish a continuous even temperature, care should be exercised to avoid pressure on the stomach. During the night a moist Priessnitz bandage may be allowed to remain in place (see page 250).

The diet for the first ten to fourteen days consists chiefly of milk, neither hot nor cold. During the first two or three days of the fourteen, a quarter of a liter ($\frac{1}{2}$ pint) of milk is given per day, in tablespoonful doses at regular intervals. This quantity is then gradually increased to one-half liter, and at the end of the first

week's treatment to one liter. The caloric value of this small quantity of milk may be enhanced by the addition of cream; the increase in caloric value can be estimated from the following calculation by Strauss:

		Calories.
A	100 Gm. full milk	70
B	75 Gm. full milk + 25 Gm. cream	115
C	50 Gm. full milk + 50 Gm. cream	185
D	25 Gm. full milk + 75 Gm. cream	205
E	100 Gm. cream	250

Therefore there are present in one-half liter (1 pint) of each of these—milk, milk and cream, and cream—the following:

	Calories.
A	350
B	575
C	925
D	1025
E	1250

Yolk of egg may be added to the milk. When milk is ill-borne or patients exhibit a dislike for it, it may be made more palatable by the addition of tea, cocoa, vanilla, or milk rice and milk jellies. Beaten cream or cream jellies may be given. Milk soups with rice, oatmeal, or the infant flours (half a tablespoonful of flour to half a pint of milk) will be found agreeable to most patients. Sugar may be added to suit the taste.

When aversion to milk is very pronounced, do not insist on its use. In such cases the most suitable substitute during the first days of treatment is yolk of egg beaten up with sugar so that the patient takes two to four eggs per day; or flour soups with the addition of butter may be given instead.

When the quantity of food taken is too small, on account of severe pain, it is advisable to add to the soups such concentrated foods as plasmon or fluid somatose. Jellies made from chicken, meat or raspberries may be employed with advantage. Patients who are fond of sweets should be given syrupy fruit juices, such as are made from apples or raspberries; or malt extract may be added to the milk or cocoa.

This strict diet, as outlined, is continued for at least ten days. If the pains subside rapidly the diet may be increased. When, however, the pain persists, it is necessary to prolong the period of physiologic rest to fourteen days.

As might be expected, patients on such a regimen decrease in weight. The loss of weight, however, may be accepted calmly, since the meager diet has contributed to the comfort of the patient and shielded the gastric mucosa from undue irritation.

Near the end of the second week, if the patient's condition permits, bouillon or soups enriched with yolk of egg, breast of chicken, or squab, enter into the dietary. The flour soups mentioned may

be continued. When the pains have wholly disappeared a careful trial may be made of a few teaspoonfuls of very finely chopped breast of chicken or squab. If this is easily borne, light egg dishes are added to the dietary. Then, tentatively, a few dessertspoonfuls of mashed potatoes, softened biscuits (crackers) or zwieback may be administered. Owing to the preponderance of liquid nourishment, patients do not experience much thirst during the first and second periods of the treatment; thirst may be allayed by small pieces of ice dissolved in the mouth. The white of an egg mixed with 200 Cc. (3 vi) of water sweetened with a teaspoonful of sugar is recommended as a beverage.

The diet during the second period (beginning with the fifteenth day of the treatment) is maintained until the end of the third week, during which time the patient is kept at rest in bed. Carlsbad water is continued, likewise the hot applications over the epigastrium. At the end of three weeks the patient may be placed upon a more extended diet; such articles of food as light cheese, boiled chicken, squab, small steak, sweetbreads, and minced veal cutlets, are permitted. Ham and uncooked meat must be avoided. At this period of the treatment fish, such as pike or trout, well cooked and served with butter balls and butter sauce, may be introduced; also mashed potatoes, as well as other kinds of vegetables in the form of purées. The quantity of biscuits and zwieback may be increased, care being exercised that such articles are completely broken up and taken in a soft, moist condition. The milk diet is meanwhile continued.

The hot fomentations need not be resorted to so frequently during the fourth week. During the latter part of this period the patient is allowed to remain out of bed and the hot applications are discontinued altogether. The diet is arranged on an increasingly liberal basis. Such foods as biscuits, zwieback and white bread toast must be carefully masticated. The regular diet to which the patient has been accustomed is not to be resumed under two months from the initiation of the treatment.

Summary of Leube-Ziemssen Treatment. There are four cardinal points to be observed:

1. Rest in bed for one or two weeks. This relieves the pain and promotes healing. After the tenth day the patients lie down two hours after dinner.
2. Carlsbad water, a quarter-liter ($\frac{1}{4}$ pint), lukewarm, every morning for two weeks.
3. Application of a hot poultice or thermophore to the epigastrium. The poultice must be changed every fifteen minutes and kept very hot. Leube never uses poultices in the treatment of bleeding ulcers, as they are apt to cause a recurrence of the hemorrhage. During hemorrhage ice-bags are used instead.
4. Light diet of high nutritive value and ready digestibility.

Confining the patient to bed, with rectal alimentation (see page 243), for several days before beginning the Leube treatment, is helpful. The reasonableness of this procedure is apparent, since absolute rest brings about a cessation of pain and vomiting and facilitates the healing of the ulcer. Persons subject to ulcer should lead abstemious lives in regard to diet and beverages, and those who have been cured should not undertake heavy work or violent exercise within a year from the cessation of the symptoms.

Lenhartz Treatment.—Among the more recent methods of treatment of gastric ulcer, especially when complicated with hemorrhage, is that devised by Lenhartz. The principle underlying this treatment involves the maintenance of enforced nutrition from the beginning—that is, from the time of the hemorrhage. Lenhartz administers the minimum quantity of food with maximum caloric value. He argues that in the Leube treatment the nutrition of the patient is so far below his needs that the anemic condition is bound to become more pronounced and the chances for the ulcer to heal are greatly lessened. Lenhartz by his protein regimen aims to counteract the hyperacidity so frequently present in gastric ulcer. Strong emphasis is placed upon the importance of physical rather than physiologic rest of the stomach.

The Lenhartz method of treatment is as follows: Absolute rest in bed for at least four weeks. All mental excitement must be avoided. An ice-bag is placed over the region of the stomach and kept there almost continuously for two weeks; this prevents gaseous distention and promotes contraction of the walls; it also obviates hemorrhage, and eases the pain when pain is present. On the first day, even when hematemesis has occurred, the patient receives 200 Cc. (̄vij) of iced milk, in teaspoonful doses, and two raw eggs—within the first twenty-four hours. At the same time bismuth subnitrate is given twice or three times a day, 2 Gm. (30 grains) at a dose, and this is continued for ten days. The eggs are beaten up entire (with a little sugar), and the cup containing them is placed in a dish filled with ice, so that they remain cold. This food at once “binds” the supersecreted acid and therefore rapidly mitigates the pain; and the vomiting, which is often quite troublesome, ceases. The portion of milk is increased each day by 100 Cc. (̄iij), and one additional egg is given, so that at the end of the first week the patient is receiving 800 Cc. (1½ pints) of milk and eight eggs daily. Both these foods are continued in the same amount for another week. No more than a liter (quart) of milk a day is allowed at any time. Besides milk and eggs, some raw chopped meat is given between the fourth and the eighth day, usually on the sixth—35 Gm. (̄ix) in small divided doses (stirred up with the eggs or given alone); the day after, 70 Gm. (̄ij); and later, possibly more if the previous portions have been well digested. The patient is now able to take some rice, well cooked,

and a little softened zwieback. During the third week a mixed diet is tolerated, the meat being given well cooked or lightly broiled.

Among the advantages of the Lenhartz method of treating gastric ulcer are: The avoidance of partial fasting, so distressing to many patients; the maintenance of body weight; and the rapidity with which the hemoglobin attains the normal after hemorrhage of greater or less severity. On the eighth day after a hemorrhage Lenhartz sometimes prescribes, in addition to bismuth, Bland's iron in finely powdered form.

DIET IN ULCER OF THE STOMACH (LENHARTZ).

Days after hemorrhage.	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Eggs	2	3	4	5	6	7	8	8	8	8	8	8	8	8
Milk (Ct)	200	300	400	500	600	700	800	900	1000	1000	1000	1000	1000	1000
Sugar to the eggs (Gm.)			20	20	30	30	40	40	50	50	50	50	50	50
Raw beef (Gm.)						35	35	35	35	35	35	35	35	35
Milk rice-ground rice (Gm.)						100	100	200	200	200	200	300	300	400
Zwieback (Gm.)								20	40	40	60	60	80	100
Raw ham (Gm.)									50	50	50	50	50	50
Butter (Gm.)									20	40	40	40	40	40
Calories	280	420	637	777	955	1135	1588	1721	2138	2478	2941	2941	3007	3073

Sippy Treatment.—*Neutralization of Free Hydrochloric Acid in the Stomach.*—B. W. Sippy states that after an experience of twelve years in the treatment of gastric and duodenal ulcer on the principle of protecting the ulcer from corrosion by the gastric juice, he is convinced that the vast majority of cases now treated surgically could be readily cured by the protective method. The reason why a gastric ulcer does not heal as rapidly as an ulcer located elsewhere in the body is that its granulating surfaces are periodically subjected to the digestive action of the gastric juice. Sippy's method consists of hourly feedings, and the administration of alkalis between the feedings. A wide variety of soft and palatable foods is permitted, the basis of the diet being milk, cream, eggs, cereals, and vegetable purée. The alkalis prescribed are calcined magnesia (heavy), sodium bicarbonate, and bismuth subcarbonate. The antacid effect of the magnesia is greater and more prolonged than that of sodium bicarbonate, but its too free employment is apt to result in diarrhea. The bismuth has, of course, an astringent effect. The alkali prescriptions alternate—first magnesia and sodium bicarbonate, then bismuth subcarbonate and sodium bicarbonate. As much as may be required to neutralize the acidity is administered. If there should be excessive hydrochloric acid secretion at night, this is removed with the stomach tube each night until the irritability of the gastric glands has subsided. The patient remains in bed three or four weeks. The results obtained by this method are, in the words of the writer,¹ "almost beyond belief."

¹ B. W. Sippy: Gastric and Duodenal Ulcer. *Journal of the American Medical Association* May 15, 1915.

I make use of the following diet in the manner outlined below:

DIET.

Soft or hard-boiled eggs.
Milk.
Bread.
Milk toast.
Crackers.
Strained cereals with cream and sugar.
Rice.
Custard.
Blanc-mange.
Junket.
Plain ice-cream.
Mashed or baked potato with cream or butter.

8.00 A.M. Breakfast consisting of food from the above diet list
9.00 A.M. Alkali powder.
10.00 A.M. Two ounces of a mixture of half milk and half cream
11.00 A.M. One soft or hard-boiled egg
12.00 M. Noon meal consisting of food from the above diet list.
1.00 P.M. Alkali powder
3.00 P.M. Three ounces of a mixture of half milk and half cream.
4.30 P.M. Alkali powder
6.00 P.M. Evening meal consisting of food from the above diet list
7.00 P.M. Alkali powder
9.30 P.M. Tumbler of half milk and half cream, one hard-boiled egg, white bread and butter

If in pain during the night, take the alkali powder.

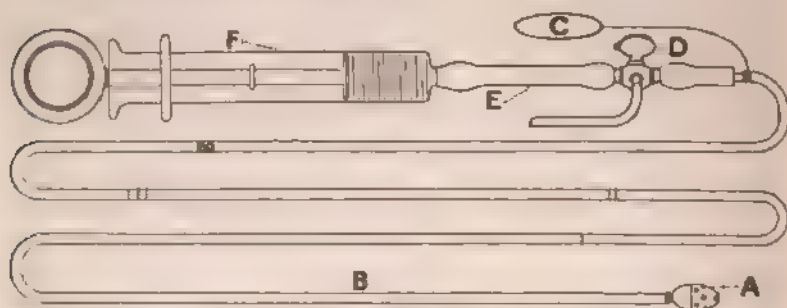


FIG. 85. Einhorn duodenal tube. A, metal capsule, the lower half provided with numerous holes, the upper half communicating with tube B. I II III, marks of 40, 50 and 70 centimeters from capsule, C, rubber band with silk thread attached to end of tubing which can be placed over the ear of the patient, F, feeding syringe, E, flexible connecting tube, D, three-way stopcock.

Einhorn's Duodenal Alimentation.—Einhorn has devised an instrument, his so-called duodenal tube, by which food can be introduced directly into the duodenum (Fig. 85), thus not only sparing the stomach the work of digestion, but, what is of more consequence, avoiding the accumulation of free hydrochloric acid in this organ. A small metal capsule (14 mm. long, 23 mm. in circumference), perforated, is attached to a long thin rubber tube (8 mm. in circumference and 1 meter long) which is connected at the other end with a feeding syringe (Fig. 85).

Feeding is begun just as soon as there is no longer any doubt that the end of the tube has passed beyond the pylorus. This can be easily ascertained by aspirating some of the fluid with the syringe. If the contents are clear, this indicates gastric juice, while yellow or greenish-yellow liquid indicates duodenal contents. The food should be introduced very slowly, always at body temperature, and at two-hour intervals. After each feeding, water should be forced through the tube, and afterward a little air, to expel the contents of the pump into the duodenum, after which the stopcock attachment of the tube is closed. The tube may remain in the digestive tract for ten to fifteen days without causing appreciable irritation or discomfort to the patient.



FIG. 86.—Duodenal feeding. (Einhorn.)

Einhorn's diet unit in duodenal alimentation consists of 240 Cc. (3viij) of milk, one raw egg, and 15 Gm. (3ss) of sugar of milk, well beaten. This amount is administered at a single feeding. Boiled milk is not coagulated by the secretions in the duodenum. Therefore it should be used in all feedings with the duodenal tube. In this manner the plugging of the tube by coagulated milk, which would occur if raw milk were used, is avoided. When it is desired to introduce a greater quantity of water into the system than that taken during the feeding process, a liter (quart) of physiologic salt solution may be given by proctoclysis (see page 239).

The patient may be fed while in the sitting posture, as illustrated in Fig. 86, or lying down.

Method of Procedure.—The capsule of the duodenal tube and the lower part of the rubber tube are moistened with warm water and placed in the pharynx of the patient. Then the patient drinks a little water, and the instrument soon passes into the s

To be certain that the capsule does not stick in the esophagus it is well to have the patient shake his abdomen, when a syringeful of chyme can be aspirated if the capsule is in the stomach. Now we pass a syringeful of water and then one of air through the instrument. The stopcock is then closed and the apparatus left untouched for about an hour. The patient is told not to close his mouth too tightly, lest the tube be retarded in its wanderings. He must also avoid intentional swallowing. Through the peristalsis of the stomach the capsule is pushed on farther, and usually passes through the pylorus into the duodenum and later into the upper part of the small intestine. It is advisable to have the patient read some light literature in order to divert his attention. After one hour we examine how far the capsule has progressed; if the mark III (indicating 70 centimeters from the capsule) is near the lips or inside the mouth, we try to aspirate. If the capsule is in the duodenum, we usually obtain a clear golden-yellow or watery liquid, of alkaline reaction and somewhat viscid consistency. If, however, it is in the stomach, we obtain an acid liquid resembling that first removed. This can, of course, occur if the tube lies coiled up in the stomach. Should the aspirated material be acid, we must withdraw the tube, after putting water and air through it, as far as the mark II (56 centimeters). The tube is then again closed, and after one-half to one hour the test procedure is repeated. The capsule in nearly all cases enters the duodenum on the first trial. After having fed the patient for ten to fourteen days the tube is closed and slowly withdrawn. When the esophagus is reached by the ascending capsule the patient is told to swallow, and during this act the capsule is withdrawn. Both gastric and duodenal ulcers can be healed by means of duodenal alimentation.

The Morgan Modification of Einhorn's Duodenal Alimentation.—Morgan has suggested substituting for the foregoing the Murphy drip method (see page 239) in duodenal feeding. He attaches to the upper part of the duodenal tube, by means of an additional section of rubber tubing, a porcelain-lined irrigator of 500 Cc. (1 pint) capacity. The irrigator is placed at such a height that it requires about an hour for 300 Cc. of fluid to run through into the gut. He begins by giving 90 Cc. of the milk, egg and lactose solution every two hours, and gradually increases so that by the end of the first day the patient is able to take the 300 Cc. at one time with perfect comfort. Morgan's patients have experienced no inconvenience from the continuous presence of the duodenal tube *in situ*, and the feedings have frequently taken place while they were sleeping and entirely without their knowledge. In addition to the feeding, Morgan gives 500 Cc. of normal salt solution per rectum, by the drop method, thus adding to the body fluids and keeping the feces soluble and the bowel actions regular.

Medicinal Treatment.—By the administration of drugs in the treatment of gastric ulcer an endeavor is made to stimulate cicatrization, to cover and protect the ulcer from chemical irritation, and to neutralize the gastric acidity, whether due to the normal acid or to any of the abnormal acids of fermentation.

In a large percentage of cases of gastric ulcer, pain can be stopped by the administration of sodium bicarbonate in large dosage. The quantity required to overcome the hyperacidity and diminish the pain is large, usually 10 to 15 Gm. (3iiss-iv) a day. The best way of giving the medicine is to dissolve a teaspoonful in lime-water, add a little spirit of peppermint, and have the patient sip the solution teaspoonful by teaspoonful until the pain disappears. The following prescription is serviceable:

	Gm. or Co.	Mxv
R—Spiritus menthae piperitæ	1 0	3ss
Magnesiæ oxidi	2 0	3i
Sodii bicarbonatis	4 0	5ss
Cretæ præparatæ	2 0	

Misce.

Sig—Stir a teaspoonful in half a tumbler of water, and sip slowly, a teaspoonful at a time, until the pain is relieved.

The proportion of chalk and magnesia will vary in accordance with the tendency to diarrhea or constipation on the part of the patient. Sodium bicarbonate by itself has the objectionable feature of easily forming sodium lactate, sodium chlorid, and other purgative salts. This inconvenience can be overcome with the aid of chalk or opium. The formation of sodium chlorid is a grave fault, as this salt is a constant source of hydrochloric acid in the presence of gastric juice. To overcome this objection sodium bicarbonate should always be combined with other alkalis or inert powders which may, in part at least, prevent the formation of sodium chlorid. The following combinations are in use; each is for one powder, which may be repeated four or five times a day. They may be given in alternation, every hour, in the endeavor to neutralize the hyperacidity, thus favoring the healing of the ulcer.

	Gm. or Co.	gr. xv
R—Sodii bicarbonatis	1 0	gr. xij
Calci carbonatis	0 2	gr. v
Bismuthi subnitratæ	0 3	

Misce.

	Gm. or Co.	gr. x
R—Sodii bicarbonatis	0 6	gr. iiij
Calci carbonatis	0 2	gr. v
Pulveris talci	0 3	gr. vi
Bismuthi salicylatæ	0 4	

Misce.

	Gm. or Co.	gr. viij
R—Sodii bicarbonatis	0 50	gr. iv
Cretæ præparatæ	0 25	gr. iv
Bismuthi subnitratæ	0 25	gr. ½
Pulveris opii	0 01	

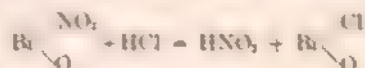
Misce

	Gm. or Co.	gr. x
R—Sodii bicarbonatis	0 60	gr. iiij
Magnesiæ oxidi	0 20	gr. iiij
Pulveris talci	0 20	gr. ½
Pulveris belladonnæ radicis	0 02	

Misce.

Bismuth preparations were employed over a century ago by Olier as a panacea for spasm of the stomach. The use of bismuth in the treatment of gastric ulcer was suggested about the middle of last century by Budd and Trousseau. It remained, however, for Fleiner, following the advice of Kussmaul,¹ to bring the bismuth treatment impressively to the notice of the profession. The method which is strongly advocated by Fleiner is as follows: The stomach tube is discarded entirely, and bismuth suspensions (24 to 5 drams of bismuth subnitrate in a tumbler of warm water) are taken by mouth, in the morning, on an empty stomach, the latter having been cleansed three-quarters of an hour to an hour previously with about 150 Cc. (5 ounces) of Carlsbad or Vichy water. The bismuth is given daily, the dose being more or less rapidly increased, reduced, or discontinued, according to the clinical course. Breakfast is taken after half an hour's rest. The bismuth treatment should be employed daily at the beginning; later, every other day or every third day. It should be continued as long as necessary (see page 265).

Bismuth subnitrate is a salt formed by the combination of bismuth with nitric acid. Nitric acid is caustic, antiseptic, and astringent. Bismuth subnitrate is insoluble in water and passes quite well through the stomach into the duodenum without much change. It has been proved by the Roentgen ray that in the presence of ulcer some of the bismuth adheres to its raw surfaces. While adhering, the subnitrate disintegrates slightly and liberates some of its nascent nitric acid, which acts locally as a stimulant, astringent, and antiseptic. The nascent nitric acid coagulates the albuminous surface of the ulcer, which thus acts as a protective during the time of healing. If the practitioner has this object in view, he should not prescribe bismuth subnitrate with an alkali, for the alkali would destroy the small quantity of nascent acid developed. It is impossible to secure as good a result in the treatment of gastric ulcer with any other salt of bismuth as with the subnitrate. The inefficiency of bismuth subcarbonate is due to the absence of nitric acid, in the decomposition of the subcarbonate, carbon dioxide is evolved. The chemical action of bismuth subnitrate within the stomach and duodenum is (1) antisecretory, (2) astringent, and (3) antiseptic. For the better understanding of these three bismuth effects, it is necessary to appreciate the chemical process which takes place upon the disintegration of the bismuth in the hydrochloric acid gastric contents. The decomposition of bismuth subnitrate takes place according to the following formula:



¹ Charles D. Axon: The Healing of Gastric and Duodenal Ulcers with Bismuth, *American Journal of the Medical Sciences*, October, 1912.

It has been found that the effective constituent is not the bismuth oxid thus formed, but the simultaneously liberated nitric acid; the three chemical effects above mentioned are due to this factor. I have used large doses of subnitrate of bismuth in the treatment of gastric and duodenal ulcer for over twenty years, without one case showing symptoms of ill effect. I prescribe it in aqueous suspension only, to be taken before meals, three times a day. The bismuth should be continued for one to four weeks during the cure. I give it as in the following prescription:

	Gm. or Cc.	
R—Bismuthi subnitrat ^{is} , c. p.	60 0	3ij
Aque destillat ^e q. s. ad	240 0	3viij
Misce.		
Sig.—Shake well. Tablespoonful three times a day, before meals.		

Nitrate of silver has been used for a long time in the treatment of gastric ulcer. Johnson, who was the first to recommend it, had observed that in his cases of epilepsy all the gastric symptoms disappeared after the administration of nitrate of silver. I have seen many cases in which all gastric symptoms caused by ulcer cleared up after a course of nitrate of silver, and found that the silver salt acts favorably in cases in which pain is present when the stomach is empty. It probably has the effect of an antacid in such cases.

	Gm. or Cc.	
R—Argenti nitratis	0 3	gr. v
Aque destillat ^e	180 0	3vj
Misce.		

Sig. —A tablespoonful in a wineglass of distilled water, three times a day, half an hour before meals.

Care should be exercised in the administration of silver salts, lest the condition known as argyrisms result from their too long-continued use (see page 267).

Chronic ulcer of the stomach may keep up a continuous irritation of the vegetative nervous system (see page 387). This may induce symptoms of a secondary neurosis manifested by pylorospasm, hyperacidity and hypersecretion. We should aim to keep the stomach free from sensory irritation in order to prevent spastic contraction, and endeavor to promote local circulation. Atropin combats the neurosis by inhibiting the impulses through the vagus, and many cases are successfully treated with this drug (see page 435). Atropin sulphate should be administered morning and evening for four to six weeks, in the dosage of 0.001 to 0.0015 Gm. ($\frac{1}{16}$ to $\frac{1}{8}$ grain) hypodermically (see page 271).

Scarlet red has been used for the healing of gastric ulcer. The dyestuff is not poisonous and can be given without deleterious effect. It has a stimulating action on epithelial cell proliferation. Scarlet red 0.5 Gm. ($7\frac{1}{2}$ grains) in capsule three times a day, before meals, or 1 per cent. in olive oil, may be employed.

I have used tincture of iodine, a five-drop dose in a wineglass of water to be taken three times a day on an empty stomach. This treatment has allayed the pain and put the patient at ease when other therapeutic agents were ineffectual.

Olive oil, owing to its high nutritive value and its absolutely unirritating properties, is a therapeutic agent worth a careful trial. It has a decidedly restraining action upon hydrochloric acid secretion. In recent cases of ulcer several spoonfuls of the oil may be administered daily, the patient rinsing the mouth with some good mouth-wash each time after taking the dose. The quantity of oil is gradually increased up to 150 Cc. (3v) per day, taken in three portions. There is nothing to prevent giving nutrient enemata, since the oil, as a rule, does not cause diarrhea, though it usually relieves the constipation from which patients with gastric ulcer are apt to suffer. Generally in eight days the digestive trouble disappears, but it is wise to continue the oil, associated with an appropriate diet, for two weeks longer. This treatment is particularly efficacious in chronic ulcer of the stomach, even when surgery proves of no avail (see page 271).

For the treatment of hemorrhage from the stomach, see Chapter XXVI on Gastric and Intestinal Hemorrhage.

Treatment by Antilytic Serum.—Antilytic serum has recently been employed with some success in the treatment of gastric and duodenal ulcer. The serum of a healthy individual contains, in addition to its other constituents, a substance which stimulates the repair of tissue cells and limits cell destruction by antagonizing certain enzymes of fixed and wandering cells. This substance is of the nature of antitrypsin and is attached to the albuminous portion of the serum; it has been called by Hort antilysin. Antilytic serum recommended for use is the normal blood serum of the horse, fresh, atoxic and sterile, in the natural condition or with its antilytic valency increased by the addition of globulin-free serum. The treatment is applicable to cases of gastric ulcer with or without hemorrhage. Complete rest in bed for two or three weeks must be insisted upon.

The antilytic serum is administered by mouth three or four times a day, immediately after meals, each dose in half an ounce of water. If pain is severe, 60 to 80 Cc. (3ij-iss) is given in divided doses in the twenty-four hours. In all severe cases the serum treatment should be continued for six weeks. Marked relief from pain has been experienced within twenty-four hours after the beginning of the treatment.

Treatment by Bacterial Vaccines.—The treatment of gastric and duodenal ulcer by means of bacterial vaccines was suggested to the author by the work of Turck on the experimental production of gastric ulcer. Recently Rosenow has been able to produce in the lower animals, by intravenous injection of living streptococci

resembling those found in rheumatic fever (*Streptococcus viridans*), ulcer of the stomach and duodenum. The ulceration is due to a localized infection and secondary digestion. The ulcers thus produced are usually single, clean, deep, bleed easily, and resemble the human gastric ulcer in many respects. In some of the experiments cholecystitis was induced, with the beginning formation of gallstones. Appendicitis was also found. This experimental study is valuable from a therapeutic viewpoint when we consider the opsonic work of Sir A. E. Wright and his vaccines made of killed bacteria.¹ It would seem rational to use *Streptococcus viridans* vaccine in the treatment of gastric ulcer. Autogenous bacterial vaccines, or vaccines sensitized by homologous serums, may be used.

Surgical Intervention. The indications for surgical intervention are as follows: (1) In sudden severe gastric hemorrhages threatening life. The ulcer should be excised in the interval. If it is possible to exactly locate the seat of the ulcer without endangering the condition of the patient through too much handling of the stomach, operate at once. (2) Often-repeated small losses of blood that cannot be checked, when the patient becomes anemic and unable to take sufficient nourishment. (3) In acute perforation, when the prognosis depends upon an early recognition of the condition and immediate operation as soon as the diagnosis has been made. (4) An ulcer situated at the pylorus, with ensuing stenosis, stasis, decomposition of the stomach contents, and derangement of the gastric mechanism. (5) In patients who suffer from pain, disturbance of digestion, vomiting, anemia, melancholia, inability to work, and general nervous break-down. (6) Gastric ulcers with formation of tumor, no matter where the location may be, always demand excision of the tumor.

The value of gastroenterostomy in gastric ulcer is dependent upon the situation of the ulcer; the nearer it is to the duodenum the better the prognosis. Operation, which under certain circumstances is extremely grave, should be considered carefully and in all its aspects before being decided upon. Medical measures can bring about relief and perhaps cure in a large proportion of cases, particularly acute ones. The possibility of chronic ulcer degenerating into carcinoma must be taken into consideration in summing up the indications for surgical intervention. It is better when possible to excise chronic ulcers, because there is always a possibility of later carcinomatous development. In a study of one thousand cases of gastric carcinoma, by Friedenwald, 7.3 per cent. gave a direct history of ulcer. When an ulcer is beginning to undergo carcinomatous change, the hyperacidity and secretion of gastric juice diminish.

¹ Charles D. Aaron, Observations of Opsonic Therapy, New York Medical Journal, December 1, 1906.

A simple uncomplicated gastric ulcer is not, in my opinion, a case for surgical intervention. Only in the event of complications, or in ulcers which defy thorough internal treatment, impairing nutrition by interference with motility, is there any indication for surgical treatment. The fact should always be taken into consideration that in the present state of the art of diagnosis we can have only a suspicion as to the seat of the ulcer. We know that four-fifths of all gastric ulcers are situated at the lesser curvature on the posterior wall of the stomach a surgically inaccessible place. Unless, therefore, there is a well-developed ulcer of the pylorus, which has been diagnosticated by the signs of retention, it is impossible to make a safe prognosis of recovery or even of improvement through surgical means.

So far as surgery is available, no procedure but removal of the ulcer by excision or gastroenterostomy is to be considered. However, excision of the ulcer does not remove the cause nor the tendency to re-formation; nor does it improve motility. Neither does it reduce hyperacidity; but it does remove the dangers accompanying the ulcer, such as hemorrhage, perforation, and malignant degeneration. Gastroenterostomy and favorable drainage protect the ulcer from irritation by the hyperacid gastric contents, and some ulcers which have defied every kind of treatment will heal or become latent after this operation. Ulcers of the pylorus or duodenum can be cured by gastroenterostomy, but no others.

It is always necessary to pay special attention to the diet after stomach operations in order to achieve the most favorable results. It is certainly surprising to observe that a patient, compelled for years to live on milk, broth, and soups, is allowed at once to partake of roast beef and potatoes. It is an overestimation of surgical effect to suppose that a stomach which has been seriously impaired for a number of years can suddenly develop normal function. It is irrational to allow such a patient to get out of bed after a couple of weeks and to discharge him as cured at the end of three weeks. After the operation a careful dietary should be maintained for weeks and even months; the surgeon should be assisted in the care of such convalescents by an internist. This course, together with the simultaneous use of alkalis, affords the best security against recurrence, especially at the jejunum, in which location ulcer is apt to develop as a sequela of gastroenterostomy.

When dyspeptic symptoms are persistent, following the surgical operation of gastroenterostomy, it has been found that duodenal alimentation (see page 500) gives most gratifying results. Adhesions, or small ulcers of the stomach or the jejunum in the vicinity of the new stoma, are the most frequent conditions causing the new disturbances. Duodenal alimentation should always be carried out before submitting the patient to further surgical measures.

Complications.—One of the most frequent complications of gastric ulcer is hemorrhage. Such hemorrhages can usually be stopped by internal measures (see Chapter XXVI), and, if these should fail, operative intervention is not likely to help (see page 520).

Perforation in Gastric Ulcer.—Statistics show that the site of gastric ulcer is, as a rule, on the posterior wall of the stomach, yet perforations occur most frequently from ulcers on the anterior wall. They break by sloughing through the anterior wall directly into the peritoneal cavity. A sudden severe burning pain in the epigastric or umbilical region is often the first symptom of a perforation. The pain is characteristic, inasmuch as it never shoots from one part of the abdomen to another, but remains localized. Frequently it is so severe as to compel the patient to cry out, and is often followed by collapse, sudden pallor, a quick, feeble pulse, cold, clammy skin, and anxious countenance. The passage of air from within the stomach into the peritoneal cavity will immediately produce an effect on the sympathetic nerves, resulting in shock, when, owing to the obtunded senses, pain disappears. Acute pain, fall of temperature, rapid pulse, vomiting, tenderness in the epigastrium, rigidity and shock, demand immediate surgical intervention. The operation may be very simple for perforation at the greater or lesser curvature or at the anterior wall of the stomach. If the perforation has taken place at the posterior wall the operation is most difficult and usually does not do any good.

The statistics in perforation show such unfavorable results from internal treatment that it seems imperative to resort at once to surgery unless there are very important considerations to contraindicate it. About one-half of the ulcer patients who have successfully gone through an operation for perforation remain quite well for years, fully capable of work, and practically free from gastric symptoms. Only a small proportion develop severe ulcer symptoms.

Subphrenic Abscess.—Subphrenic abscess following perforation should likewise be operated upon as soon as possible. The most frequent cause of such suppuration is gastric ulcer; it is rare to find it following any other disease. The abscess may also be subhepatic or retrocolic.

Pyloric Stenosis. The surgically most important complication of gastric ulcer is benign pyloric stenosis with subsequent dilatation of the stomach (see Chapter XXIV). The operations for the relief of benign obstruction of the pylorus are: pyloroplasty, gastrojejunostomy, and gastroenterostomy. The method and selection of the operation will depend upon the conditions at the time of operation.

Hypertrophic Stenosis of the Pylorus.—Hypertrophic stenosis of the pylorus has been successfully operated upon in very young

children. As experience in these cases accumulates we find, however, that internal treatment is often efficient and surgical intervention not often required. An important point to remember this connection is that we do not know how the operative result accomplished in young children will regulate itself in advance years.

During the past few years it has been found that the Ramstedt operation is the one generally adopted. This consists in incising in a longitudinal direction the thickened and hardened pylorus through the muscularis down to the mucosa without opening the latter.

In congenital hypertrophic stenosis of the pylorus, a child at birth seems well nourished, but soon begins to vomit its food. The quantity of vomited material increases from day to day; a alteration of food, modified or peptonized milk, seems to have little or no influence on the vomiting, which continues regardless of the quantity of food taken. By the use of the stomach tube we find that if there is no vomiting the food taken is retained in the stomach a long time. The weight of the child, meanwhile, continues to decrease and the little patient looks old and wrinkled. Constipation is usually present. The tongue and mouth are moist and clean. Upon inspection the abdomen is found to be flat, and a peristaltic wave can be seen to pass over the stomach. Frequently the stomach contents may be outlined through the abdominal wall and the visible waves of peristalsis easily made out. An epigastric tumor points to pyloric stenosis.

A rare condition found in babies is due to an infection through the omphalomesenteric duct and urachus that induces adhesion of the liver and duodenum, pulling the pylorus up and kinking it in such a way as to give symptoms of pyloric stenosis.

CHAPTER XXVI.

GASTRIC AND INTESTINAL HEMORRHAGE.

ANY different conditions may cause gastric or intestinal hemor-

rhage. Bleeding in the stomach may take place without any symptoms. This can also be said of hemorrhage in the

intestine. Unless patients complain of hematemesis or melena, they should consult a physician until signs of intense anemia appear.

Diagnosis. The diagnosis of gastric hemorrhage is usually not a difficult matter. There is, as a rule, hematemesis, accompanied by melena. Hemorrhages from the mouth and respiratory tract, in which the blood has been carried to the stomach by swallowing, must be excluded. Due inquiry must be made as to possible prior and causative injuries to the head, or recent affections of the lungs. In gastrorrhagia there are many circumstances which indicate the exclusively gastric nature of the trouble; in almost every instance there is a history of stomach disturbance, often of long duration, preceding the hemorrhage. Hemorrhage without any previous symptoms is

unusual. Furthermore, the history and clinical symptoms render diagnosis fairly easy as to the particular disease causing the hemorrhage, whether gastric ulcer, superficial ulceration, capillary ang. or carcinoma. Gastric hemorrhage may occur in the following conditions: Venous or varicose hemorrhage in cirrhosis of the liver or obstruction to the portal circulation; parenchymatous gastric hemorrhage in diseases of the heart, liver, or blood; and chronic gastritis; stenosis of the pylorus; miliary aneurysm; injury from foreign bodies in the stomach; caustic poisons in the stomach; syphilis; arteriosclerosis; septicemia; rupture of vessels or of an aneurysm of neighboring structures into the stomach; anemia and disturbances of menstruation (vicarious intestinal hemorrhage); hemorrhoids; neurogenous disturbances (epilepsia, gastric crises); and cholelithiasis. Intestinal hemorrhage occurs either as severe acute hemorrhage (in which, as a rule, considerable quantities of blood are lost) or as a chronic condition during the course of which small quantities of blood are lost at different times. The mildest form of hemorrhage is that in which mere traces of blood appear in the feces. Hemorrhage may occur in cases of ulcer, stenosis, ileus, malignant tumor, disease of the liver, embolism of various sized branches of the mesenteric artery or thrombosis of the corresponding veins, and hemorrhoids.

Gastric ulcer is the most common cause of hemorrhage from the stomach, occurring in 5 per cent. of the entire population, according to Ewald and others. Hematemesis occurs in at least 40 per cent. of all cases of gastric ulcer, and many authorities place the proportion much higher. In a study of 1000 cases of gastric ulcer, Friedenwald found hemorrhage in only 22 per cent. It is fatal in 8 per cent. of the cases in which it occurs, according to the conservative estimate of Leube, and we cannot question that it is indirectly fatal in a much greater number of cases through anemia and its remote consequences.

Severance of the omental bloodvessels leads to thrombosis in them, which may extend back some distance from the point of origin. This thrombosis may occlude such vessels as the gastro-omental, gastric, and others. The breaking-off of emboli and their lodgment in the veins of the stomach and intestine frequently occurs. This is followed by necrosis and ulceration of the parts supplied by these vessels, and often by fatal secondary hemorrhage.

Differential Diagnosis.—The differential diagnosis of doubtful cases, as between gastric and duodenal hemorrhage, is a matter fraught with great difficulty. The following symptoms indicate a duodenal origin of the bleeding: (1) Pain about one to three hours after meals, which is relieved by the taking of food; (2) considerable melena, associated with hematemesis or existing alone; (3) the pain is often in the right hypochondriac region.

Prophylaxis. Only in cases in which the diagnosis is ascertained with certainty, in gastric ulcer or carcinoma in which hemorrhage may be anticipated, are prophylactic measures likely to avail. In such cases occult hemorrhages frequently precede hematemesis. The stools should be examined frequently for occult blood. Concealed gastric hemorrhage is of very frequent occurrence, not only in gastric ulcer, but more especially in carcinoma of the stomach, in which the patients often "bleed to death, as it were, by drops," without the knowledge of the physician. A very careful analysis of the feces and gastric contents in all cases in which there is the least cause for suspicion is recommended; the patient, meanwhile, should be placed upon a meat-free diet. On discovery of occult blood in the stools the patient should be placed at rest in bed; the diet should be exclusively liquid, preferably milk, and the quantity should be gradually increased until, at the end of eight days, three liters (quarts) a day are being consumed. The patient should maintain the recumbent posture until no further signs of hemorrhage are evident from an examination of the stool (see page 123).

Treatment.—*Visible (manifest) or macroscopic gastrorrhagia* is characterized by hematemesis, tarry stools (melena), or both. In the treatment of this condition the first indication is to stop the bleeding; the second, to combat the condition producing the hem-

orrhage. The patient must be placed absolutely at rest in bed, in the dorsal position. An ice-bag suspended by a frame, to avoid pressure, is placed over the epigastrium; this will have an analgesic effect and conduce to the comfort of the patient. When the patient is suffering much pain and is very restless and sleepless, morphin hypodermically will often bring immediate relief. It must be given in substantial doses to be effectual, 0.015 to 0.03 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain) every three or four hours. The addition of atropin 0.0006 Gm. ($\frac{1}{160}$ grain) relaxes the spasm. Codein phosphate 0.02 to 0.06 Gm. ($\frac{1}{4}$ to 1 grain) may be given instead of morphin.

Suppositories of extract of belladonna 0.0075 Gm. ($\frac{1}{4}$ grain) and extract of opium 0.03 to 0.06 Gm. ($\frac{1}{2}$ to 1 grain) are likewise effective. Absolute abstinence from food is necessary, thus keeping the stomach at rest not only physically but physiologically. Thirst is to be counteracted by small pieces of ice in the mouth and by rinsing the mouth with water. Proctoclysis or hypodermoclysis of physiologic salt solution is an excellent means of quenching the thirst. It is rather doubtful whether nutrient enemata should be given as a prophylactic measure against inanition from continuous hemorrhage. Nutrient enemata always cause the patient to move about and induce increased peristalsis. Bodily movements should be avoided. Instead, therefore, of nutrient enemata, proctoclysis should be employed (see page 239). When the hemorrhages are moderate in amount, nutrient enemata may be resumed earlier than in the severer cases. While the hemorrhage is in progress the ice-bag should be replaced at night by a Priessnitz bandage. Hot compresses should not be employed after recent hemorrhages.

Should the quantity of blood lost be large, normal saline must be administered by hypodermoclysis or by intravenous injection.

Treatment by Lavage.—A number of writers, among them Ewald, recommend lavage of the stomach with ice-water to remove clots and at the same time to act as a styptic.

Kaufmann believes that gastric lavage is the most expedient means in the treatment of severe hemorrhage from gastric ulcer, provided it be carefully applied. It relieves overdistention by removing the stagnating masses of accumulated blood, acid secretions, food remnants, and gas, which are usually present in such cases, and which not only give rise to nausea and pain but act as a constant source of irritation to the mucous membrane, inducing hypersecretion and thus increasing the amount of gastric contents. The removal of this material allows the emptied stomach to contract, and this aids in the occlusion of the eroded vessel. The thrombus ordinarily formed does not usually fill the opening of the bloodvessel completely. Lavage removes such inefficient thrombi and gives the bleeding vessel a chance to contract and

form a more efficient thrombus. With a carefully performed lavage there should be no danger of causing perforation by over-distention; the amount of water in the stomach at any one time should be comparatively small, and if perforation from the pathologic process should occur the cleansing of the stomach will prove beneficial, since it prevents the entrance of gastric contents into the peritoneum. It is well known that the prognosis in perforation is best when the perforation takes place at a time when the stomach is empty (see page 197).

After lavage, large doses of crystalline bismuth subnitrate should be administered. Bismuth in crystalline form is supposed to adhere more tenaciously to the surface of the ulcer than the ordinary amorphous form, so that the blood is agglutinated to the bismuth mass. Bismuth is not sufficiently astringent to contract the bloodvessels and thereby stop the hemorrhage; it does, however, aid in the coagulation of the blood, at the same time exercising a soothing influence upon the gastric mucous membrane (see page 517).

Treatment by Enemata.—Hot-water enemata have been employed with favorable results. The enema consists of $\frac{1}{2}$ liter (1 pint) of water at 120° F.; this should be given three times a day. The object is to produce reflex anemia in the upper portions of the intestine. Hot enemata promptly check intestinal hemorrhage in typhoid fever, as they do bleeding from the rectum, sigmoid, and colon (see page 220).

Medicinal Treatment.—Hemostatics.—Ergot has a direct hemostatic action and should always be given subcutaneously. The following prescriptions have been found useful:

	Gm. or Co.	
R—Extracti ergotæ	1 0	gr. xv
Aquæ destillatæ	5 0	℥jxxx
Phenolis lupulæfacti	0 06	℥j
Misce.		
Sig.—Fifteen to thirty minims to be injected subcutaneously.		

	Gm. or Co.	
R—Extracti ergotæ	2 5	gr. xxxviiss
Glycerini	5 0	℥jxxx
Aquæ	℥℥	
Misce.		
Sig.—Fifteen to thirty minims several times daily, hypodermically.		

Emetin hydrochloride has proved of great value. It acts on the smooth fibers in the bloodvessels, causing them to contract, and thus reduces the congestion and the bleeding. It may be administered hypodermically, 0.02 Gm. ($\frac{1}{2}$ grain) twice daily for two or three days.

Hydrastin hydrochloride is less effective:

	Gm. or Co.	
R—Hydrastinæ hydrochloridi	0 5	gr. viiss
Aquæ	4 0	3j
Misce.		
Sig.—Fifteen to thirty minims, hypodermically.		

The employment of gelatin is more promising. Sterile gelatin is furnished to physicians in strengths of 10 per cent. and 20 per cent. It is marketed in sealed glass tubes, ready for use, and is liquefied by placing the tubes in hot water. It is then taken up by means of a large syringe directly from the glass tube, and injected subcutaneously: 40 Cc., containing 10 per cent. of gelatin, constitutes a single dose for adults; in obstinate cases this may be repeated several times. Strict aseptic precautions must be observed. Gelatin injections do not give rise to symptoms of anaphylaxis, as serum injections sometimes, though very rarely, do. Gelatin, like horse serum, increases the globulin and promotes coagulability of the blood. The slow instillation into the rectum of a saline solution containing 1 per cent. of gelatin has been found of considerable value in intestinal hemorrhage. A combination of calcium chlorid with sterilized gelatin has been prepared under the trade name of kalzine. Subcutaneous injection of kalzine is said to give good results in hemorrhage.

The action of gelatin is supposed to be due to its lime salts; lime acts as a hemostatic. In severe hemorrhages chlorid of lime has been employed in 5 to 10 per cent. solution in the form of small rectal enemata—10 to 12 Cc. (2 or 3 fluidrams) every two or three hours. Wright recommends calcium lactate, to be administered by mouth, 1 or 2 Gm. (15 to 30 grains) three times a day. It may be administered hypodermically in the same doses.

Another preparation is stypticin, which is injected in 10-per-cent. watery solution, subcutaneously, 1 or 2 Cc. (15 to 30 minims) three times a day. Good results have been claimed for it.

Blood transfusion has been found to be an excellent hemostatic in any severe hemorrhage that cannot be controlled by the usual methods. The increase of blood coagulation and the stimulation of capillary contraction are due to active thrombin in the blood of the donor which is usually lacking in the recipient. Thrombokinase is present in the juices of all tissues. When a bloodvessel is injured, thrombokinase is secreted and unites with the thrombogen of the blood, forming the fibrin ferment to which coagulation is due. Blood or blood serum can be used to stop hemorrhage. From 10 to 20 Cc. (3iiss v) of human blood serum may be injected intravenously and the dose repeated several times. The blood can be kept in a sterile bottle in the ice-box for a few days, and the serum used when required. Human serum is safer than horse serum, since the latter may possibly cause anaphylactic reactions.

During the recent war, when blood transfusion was impossible, good results were obtained by the intravenous injection of Locke's or Ringer's solution containing 5 per cent. of acacia.

By precipitating normal horse serum, a sterile, soluble, anhydrous powder containing the fibrin ferment necessary for clotting

the blood has been obtained. This hemostatic ferment is sold under the trade name coagulose. It is readily soluble in cold water, and it possesses the great advantage, as compared with liquid serum, of retaining its coagulating principle unimpaired for long periods of time. It has been used with success in hemorrhage from the stomach and intestine.

Coagulose is supplied in 15-Cc. ($\frac{1}{2}$ ounce) glass bulbs which contain 0.65 gram (10 grains) of the desiccated powder, equivalent to 10 Cc. ($\frac{1}{2}$ ounce) of blood serum. Before being used, it is necessary to add to the powder in the bulb 8 to 10 Cc. of sterile water, the temperature of which should not be above that of the blood. The solution may be injected subcutaneously at any convenient point. One dose consists of the contents of one bulb. If the bleeding is not entirely controlled within half an hour a second dose should be given within two or three hours. In persistent hemorrhages three or four injections may be given daily for several days, and these should be continued for a short period after the hemorrhage ceases.

It has long been recognized that the formation of blood clot depends upon the evolution of fibrin from the fibrinogen of the blood, through the action of a ferment, thrombin. Thrombin does not exist as such in the blood, but is present as prothrombin, and is kept in this antecedent state by the action of a neutralizing substance, designated antithrombin. Under proper conditions the antithrombin is neutralized and thrombin released, bringing about the phenomenon of blood clot by acting upon the fibrinogen.

"Hemostatic serum" is a sterile serum derivative composed principally of prothrombin, thrombokinase and anti-antithrombin in physiologically balanced solution. It is a clear, light-amber colored liquid, and is physiologically adapted to intravenous, subcutaneous, intraspinal or intraperitoneal injection. It is indicated in the treatment of all types of hemorrhage, and is of particular value in cases depending upon faulty coagulation of the blood. The average dose is 1 to 2 Cc., given intravenously or subcutaneously. In known hemophilic cases the proper dose is 5 Cc. The injection should be repeated every four to six hours until perfect control is established.

Thromboplastin is the trade name of a solution of thromboplastic substances normally found in the brain of the ox. It is a liquid extract and its activity is said to be due to the presence of kephalin, a useful hemostatic agent. The fibrin ferment action is said to become manifest in one to two minutes, and the clot is decidedly firmer than that which occurs spontaneously. Thromboplastin may be administered by mouth in gastric and intestinal hemorrhage in the dose of 20 Cc. diluted with 300 Cc. (10 ounces) of water.

Kephalin is an ether-alcoholic extract of brain substance evaporated until the residue is a yellow lipid. It is not destroyed

by boiling. For use by mouth or intramuscular injection the dose is 10 to 30 drops in physiologic saline solution, repeated every four to sixteen hours. Its action is similar to that of thromboplastin.

By fractional centrifugalization of animal blood, a natural physiologic styptic consisting of blood platelets has been obtained; it is sold under the trade name *coagulen*. It can be dispensed in powder or tablet form, and is put up as a solution in ampoules especially for subcutaneous or intravenous injection. In giving *coagulen* intravenously the injection should be made very slowly, and discontinued at once if any headache, cardiac pain or eye derangement appears.

Extracts of the pituitary and the thyroid gland, administered in small doses, the former hypodermically, have a hemostatic effect. Epinephrin is recommended, in solution of 1 to 1000, 1 or 2 Cc. (15 to 30 drops) by mouth, two or three times, at short intervals, the day of the hemorrhage.

In collapse after profuse hemorrhage, caffeine may be given:

		Gm. or Cc.	
R—Caffeine	3 0	gr. xlv
Aqua	30 0	ʒi
Misce			
Sig—Fifteen to thirty minims, hypodermically.			

When the hemorrhage has ceased for several days and examination of the feces shows a complete cessation of occult bleeding, the administration of more copious nutrient enemata is indicated. Feeding by mouth may now be begun. The quantity of milk should be gradually and slowly increased, so that about 1 liter (quart) will be consumed on the eighth day after the cessation of the hemorrhage. When the hemorrhage is due to gastric or duodenal ulcer, the "Leube cure" or "Lenhartz cure" may be instituted at this period (see Chapter XXV).

Drugs, as a rule, play a subordinate part in the treatment of hemorrhage from gastric ulcer, especially when proper dietetic treatment can be instituted and carried out. When, however, patients must be treated while following their usual occupations, or when pains persist in spite of dietetic measures, medication proves especially valuable.

Bismuth in the form of one of its salts is employed probably more extensively than any other drug in the treatment of hemorrhage of the stomach and intestine. The bismuth salts owe their efficacy to their slightly astringent effect, which promotes granulation at the surface of the ulcer. Animal experimentation has shown that bismuth stimulates the secretion of mucus, which, together with the salt itself, forms a protective film upon the denuded portions of the gastro-intestinal mucous membrane. This covering is capable of protecting the ulcerated points from

irritation by both food and gastric juice. The bismuth meanwhile becomes oxidized, changing into the dioxid of bismuth. Under this bismuth crust the formation of granulation tissue can proceed without interruption, resulting in the so-called bismuth eschar. Since the bismuth preparations are astringent, they diminish secretion, mitigate the severity of pain, and arrest hemorrhage. A single large dose is usually effectual. Bismuth suspension, consisting of 30 grains (1 ounce) of bismuth subnitrate in 60 Cc. (2 ounces) of water, may be given (see page 504).

One of the most lauded hemostatics for the treatment of gastric hemorrhage is escalin, introduced by G. Klemperer. This is a paste of finely powdered aluminum in glycerin, and, according to Klemperer, it possesses the property of arresting bleeding more effectually than other means. It has been found that escalin stimulates the secretion of gastric juice, and the general conclusion has been that its administration cannot be considered a valuable addition to the therapeutic measures at our disposal for arresting hemorrhages of the stomach.

Silver nitrate is similar in its action to bismuth. In the treatment of conditions associated with gastric hemorrhage we begin with a solution of 0.25 Gm. (4 grains) in 120 Cc. (4 ounces) of distilled water, one tablespoonful to be taken three times a day when the stomach is empty. The strength of the solution is gradually increased to 0.3 Gm. (5 grains) in 120 Cc. (4 ounces). The stronger solution should be continued for five days. Finally, a solution of 0.4 Gm. to 120 Cc. (6 grains in 4 ounces) is taken, the dose being the same as before (one tablespoonful). In the meantime the Leube method of treatment should be followed (see page 267).

Analgesics.—Analgesic drugs, such as morphin, codein, atropin, and extract of belladonna, may be administered with bismuth powders:

	Gm. or Co.	gr. ss
R= Codeinæ phosphatis	0 03	gr. ss
Extracti belladonnæ	0 02	gr. $\frac{1}{4}$
Bismuthi subnitratæ	2 00	gr. xxx

Misce et ft. pulv. no. 1, mitte x.

Sig.—One powder three or more times a day.

Atropin has been employed with advantage in the treatment of gastric hemorrhage due to slow-healing ulcers, with hyperacidity, hypersecretion, and motor disturbances (see page 435). Subcutaneous injections of atropin have produced favorable results, though the drug is not directly hemostatic; the effect is due to its inhibitory action on the vagus. The hemorrhagic blood effused into the stomach acts as a stimulus to the secretion, and thus the coagula closing up the bleeding vessels are constantly redissolved by the gastric juice; if atropin, by suppressing the secretion, prevents the thrombotic coagula from being dissolved,

it may be said to act indirectly as a hemostatic agent. At the beginning of treatment patients are given 1 milligram ($\frac{1}{60}$ grain) of atropin sulphate hypodermically morning and night. If required, however, 3 or 4 milligrams ($\frac{1}{8}$ to $\frac{1}{5}$ grain) may be administered during the twenty-four hours. Atropin, by inhibiting hypersecretion, has a marked influence on pain. Its administration may be continued for four to eight weeks. The chief untoward effects complained of by patients are dryness of the mouth and indistinct vision resulting from the cycloplegic action of the drug (see page 271).

Chloroform-water (1:120), one tablespoonful every two hours, may be given when slight pains are present. Chloroform may be prescribed in combination with bismuth also:

	Gm. or Gs.	
R—Chloroformi	1 0	℥xv
Bismuthi subnitratii	4 0	3j
Aquæ q. s. ad	150 0	℥v

Misce.

Sig.—One tablespoonful to be taken every hour.

Cocain may be made use of in the presence of pain and obstinate vomiting. Orthoform and anesthesin are valuable analgesic remedies. The prompt effect of orthoform in relieving the pain of ulcer associated with hemorrhage has been noted (see page 270).

Lenhartz recommends the following pill in the treatment of the severe anemias resulting from gastric hemorrhage:

	Gm. or Gs.	
R—Ferri sulphatis, Potassii carbonatis	3℥ 15 0	℥ss
Tragacanthæ, q. s.		

Misce et ft. pil. no. c.

Sig.—Three pills three times a day.

Iron in this form is sometimes badly borne, which fact has led to the introduction of other iron preparations. Fersan has been described on page 188. I prefer the hypodermic method of administering iron (see page 581).

As a tonic and hematinic for the relief of the anemic and emaciated condition of the patient the following may be prescribed with great benefit:

	Gm. or Gs.	
R—Ferri sulphatis exsiccati	0 00	gr. j
Manganii dioxidi,		
Quinina bisulphatis	AA 0 1	gr. iss
Extracti nucis vomicæ	0 01	gr. i
Extracti gentianæ	0 14	gr. ij

Misce et ft. caps. no. i; mitte 1

Sig.—One capsule four times daily.

For controlling persistent intestinal hemorrhage, tincture of iodin frequently gives gratifying results:

	Gm. or Co	
R—Tincture iodii	1 0	℥ xv
Sodii iodidi	0 1	gr. ij
Aque menthae piperita,		
Syrupi simplicis	6A 20,0	3 v
Misce.		
Sig.—Teaspoonful every hour.		

Hyperechlorhydria not infrequently precedes gastric hemorrhage. To counteract this condition, bismuth subnitrate is recommended in large doses after meals, wrapped in wafers or suspended in mucilage. The following combination is of value:

	Gm. or Co	
R—Sodii bicarbonatis	0 5 1 0	gr. viii-xv
Magnesi oxidi	0 65	gr. x
Bismuthi subnitratii	1 30	gr. xx
Cretae preparata	0 25	gr. iv
Misce et ft. chart. no 1; mitte xx.		
Sig.—One powder after each meal.		

Constipation may occur with the administration of the foregoing, and under such circumstances a saline laxative would be indicated (see page 284).

Operative Treatment.—Acute hemorrhage is not a condition that lends itself to surgical treatment. It can usually be stopped by internal treatment; and if this should fail, operative intervention is not likely to help. Less than 5 per cent. of the cases die of these hemorrhages without operation. By subjecting patients to operation we expose them to further dangers to which they easily succumb; while without operation they have a reasonable chance to recover. This view is shared by a large number of experienced surgeons. With internal treatment Lenhartz reports 201 cases of gastric hemorrhage with a mortality of 3 per cent., Ewald 166 cases with a mortality of 4.87 per cent., and Wirsberg 320 cases with a mortality of 5.9 per cent. If energetic prolonged internal treatment should not be successful in checking chronic oozing of blood—as can easily be observed by daily examination of the feces with the benzidin test for occult blood—operative treatment should be advised. In hemorrhage from ulcer, either resection of the ulcer, or, where this is impossible, gastroenterostomy, should be performed. The latter operation frequently stops the hemorrhage, especially if the ulcer be situated at the pylorus. In pyloric ulcer, however, it is not the hemorrhage, but the stenosis, which renders operation necessary.

CHAPTER XXVII.

EROSIONS; PERIGASTRITIS.

EROSIONS OF THE STOMACH.

Forms.—*Acute or Hemorrhagic Erosions.*—These are small abrasions of the gastric mucosa which extend partly through this layer. They are usually multiple. They occur in the new-born; in chronic diseases of the heart or arteries; in acute infections with the pneumococcus; and in septic infection. Hemorrhagic erosions of the gastric mucous membrane are sometimes complications of chronic gastritis in its early stages.

Chronic Erosions of the Stomach.—Einhorn, who was the first to describe gastric erosions as a clinical entity, defines the condition as one in which the gastric mucous membrane becomes the seat of small superficial exfoliations.

Gerhardt says that in erosions of the stomach, as shown by section, almost the entire lower stratum of the mucous membrane is, as a rule, normal. In the epithelium of these remaining glands nothing remarkable can be discovered; at the sides the glands become longer; the first ones that are intact usually curve themselves over the defect and partly cover it. Recovery seems to take place by the simple aftergrowth of the gland remnants.

Etiology.—The exact etiology of erosions of the stomach is obscure. Einhorn reports association of the condition with hyperchlorhydria, but the vast majority of cases have been ascribed to chronic gastritis. The same factors which predispose to gastritis are sometimes associated with erosions of the stomach, but in most cases of gastritis there is no evidence of erosions. In many cases in which gastritis could be excluded, Turek found erosions of the mucous membrane. He claims also to have found them, in the same cases, in other locations—mouth, pharynx, colon; and many a so-called ulcer of the rectum presents more of the symptoms of erosion than of ulcer. In lavage of the colon, particles are found in the wash-water similar to the specimens of mucous membrane found in the wash-water from the stomach of the same patients.

There are a number of predisposing conditions. Children who have been ill-fed and those who do not appropriate the full nutrition of their food, the vascular walls losing "tone" though the body weight may not suffer, are more or less subject to erosion of the stomach. The abuse of alcohol is another predisposi-

factor. Chlorosis may play an important part in the causation of erosions. It would appear that erosions result from obstruction of the circulation to the stomach, combined with irritation of the gastric mucosa.

Symptoms.—Pain is the most pronounced symptom. This comes on after partaking of food, irrespective of the kind. The pain of erosions differs from that of gastric ulcer, inasmuch as it is not intense, never boring or cramp-like, though Pariser states that in the cases under his observation the pains were described as "unbearable suffering." It is probable that the annoying constancy of this symptom impresses the patient with a sense of great severity. Pain comes on immediately after eating, persists for an hour or two, then gradually subsides. In some cases it persists all the time, irrespective of the ingestion of food. Lavage usually dispels the pain. Frequently patients have no appetite. In some cases vomiting is one of the distressing symptoms. Control investigation of the fasting stomach, in order to differentiate erosions from gastric ulcer or from a neurosis, is necessary.

Patients lose weight at the beginning of their illness, but after that the weight is fairly constant. They present a picture of emaciation, protruding jaws and hollow cheeks, but not the cachexia which characterizes carcinoma and the severe wasting diseases. Patients with gastric erosions complain of weakness and inability to work, a feeling that is most marked directly after meals.

Diagnosis.—The most important diagnostic feature of gastric erosions is the presence in the water, after lavage, of small pieces of gastric mucous membrane. Einhorn describes them as 3 or 4 mm. long, about the same width, and of a blood-red color. Under the microscope well-preserved glands and accumulations of red blood-corpuscles may be seen. Blood is almost never found in the washings which contain membranous exfoliations. This is explained by the probability that the pieces of gastric mucosa peel off some little time before the performance of lavage. When the return water is tinged with blood, this is the result of coughing which violently contracts the stomach. It is difficult to ascertain whether the exfoliations are from the same spots day by day, or from different locations (see page 97).

Pathology.—The pathology of erosions, according to Ewald (who has studied it soon after the death of the patient), presents the following picture: "The ducts of the glands were packed full of red blood-cells, having their origin from hemorrhages on the surface of the mucous membrane, which in turn could only have come from the capillary network situated close to the free surface of the mucous membrane. They develop into little hemorrhagic erosions, small streak-like or rounded losses of substance, from the size of a millet seed to that of a pea, on which at times a blackish-brown extravasation of blood is found, together with a simulta-

neous loosening of the mucous membrane." In the majority of cases there is a decrease in the hydrochloric acid secretion. In some there is more or less profuse secretion of mucus.

Prognosis.—The course of the disease is usually prolonged, extending sometimes over several years. There are, however, intervals of improvement.

Treatment. The dietetic treatment depends upon the results of analysis of the gastric contents. The condition of the secretion determines whether the case shall be treated as subacid gastritis or hyperchlorhydria. The alkalis are indicated in hyperacidity; the vegetable bitters in cases characterized by a deficiency of hydrochloric acid secretion.

There is, as a rule, marked muscle weakness; consequently food is apt to remain longer in the stomach than is normal. Time must be given for one meal to pass through the pylorus into the intestine before a second meal is taken. It is well to advocate two meals a day, one in the morning and one at night. There may be some distress in the beginning from the loss of the noonday meal, but this is purely a question of habit, and the patient soon becomes accustomed to taking two meals daily, feeling more comfortable. With great loss of motor power dietetic measures must be observed. Chopped meat and white bread are all that is desirable in the beginning. We may gradually add to this, chicken, fish (boiled or baked, not fried), sweetbread and calf's brain. Vegetables may be added later—potatoes, squash and mashed turnips.

GENERAL TREATMENT.—The indication for general treatment is the equalization of the circulation, for which the hot bath and extension movements can be employed. The patient is placed in the bath at 105° F., and the temperature is rapidly increased to 110° to 115° F. When his skin has become reddened he is taken from the bath and rubbed with ice. The ice further stimulates circulation and reduces the temperature caused by the heat of the bath.

LOCAL TREATMENT.—The local treatment of the stomach in gastric erosions is of great importance.

1. *Nitrate of Silver.*—Pain in this condition is best relieved by lavage with a 0.5-per-cent. solution of nitrate of silver, after rinsing out the fasting stomach with lukewarm water. The silver solution should be permitted to remain in the stomach for about a minute; on its removal the lavage is repeated with lukewarm normal salt solution. This treatment may be employed every other day for ten to twelve days, or until all particles of mucous membrane have disappeared from the stomach contents (see page 267).

The following prescription has been found useful:

	Gm. or Gr.	
R—Argenti nitratis	0	25 gr iv
Aquæ destillatæ	q. s. ad	240 0 Sviij
Misce		

Sig.—Tablespoonful three times a day, before each meal

2. *The Bismuth Treatment.* This consists of lavage every other day with an alkaline solution, to dissolve mucus, and the administration of bismuth subnitrate in doses of 1 to 2 Gm. (15 to 30 grains) three times a day (before meals) (see page 265).

	Oz. or Cc.	
R—Bismuthi subnitratiss	30 0	℥j
Aquæ chloroformi q. s. ad	240 0	℥viij
Misc.		
Sig. Tablespoonful three times daily, before each meal.		

3. *Suprarenal Gland.*—Einhorn recommends desiccated suprarenal gland. He administers it every other day in powder form—about 0.2 Gm. (3 grains). When this is used the nitrate-of-silver douche is omitted.

Stockton and Jones recommend attention to the general health and advise strychnin, arsenic, malt and cod-liver oil, fresh air, sunlight, mountain climbing and other invigorating exercise, to be used appropriately.

PERIGASTRITIS.

Perigastritis is an inflammation of the peritoneal coat of the stomach. It may develop in the course of ulcer of the stomach, in two forms, namely: as a loose adhesion between the stomach and neighboring organs, whereby the former is subjected to traction; and as a tumor-like infiltration caused by the gradual advance of the ulcer toward the abdominal wall.

The local inflammation runs a latent course, and the symptoms are obscured by the more pronounced pains of the gastric ulcer. Perigastric adhesions are caused by ulceration of the stomach and duodenum, gallstones in the gall bladder or bile ducts, traumatism, malignant disease, pancreatic disease, umbilical hernia, and possibly tuberculosis and syphilis. The adhesions are usually to the pancreas, liver, or spleen. Adhesions to the anterior abdominal wall are very rare. Symptoms due to adhesions arise usually in cases where the attachment is to one of the more mobile organs, which drags on the adhesions. Liver or pancreas adhesions are usually short and broad; those to the colon or gall bladder may be long and cord-like. The shape of the stomach may be markedly or only slightly altered. The pylorus may be narrowed, or the stomach may be nearly divided into two parts—hour-glass stomach. Other effects are: dilatation by traction, and interference with motility and contracting power.

Hour-glass Contraction.—Hour-glass stomach is a condition in which the stomach is divided into two cavities. It may be either congenital or acquired. The hour-glass stomach, so-called, is caused by perigastric adhesions or gastric ulcer. The diagnosis depends in the main upon the use of the stomach tube, when by inspection one may be able to see that the fluid introduced into the

stomach through the stomach tube produces a ballooning or prominence of one part of the stomach, and that this prominence suddenly subsides and after a gurgling noise another swelling shows itself in the other part of the stomach. The Roentgen ray and the bismuth meal afford the best means of diagnosis (see Plate XV, Fig. 2).

Symptoms.—The history of the case is usually a long one, and the symptoms finally complained of are not infrequently preceded by others more characteristic of gastric ulcer or gallstone colic. Pain is the most common and characteristic symptom, and a marked feature is the fact that it is frequently confined to one locality. It is usually greatly influenced by the position of the patient, but very little by food. Violent exertion often brings on the pain; it is sometimes relieved by firm pressure or bandaging. Local tenderness is usually present. The secretion of gastric juice is normal.

Forms.—Among the varieties of perigastritis are:

1. *Local Adhesive Growths, which may or may not give rise to distressing symptoms.*—These adhesions may cause pain of greater or less severity, especially when the adhesive bands are subjected to traction by various bodily movements (walking, gymnastics) or the distention of the stomach with food. The adhesions may result in disturbing the motility of the stomach. The diagnosis is often difficult, since little or nothing can be elicited by palpation. A diagnosis of perigastritis is warranted when, after the healing of a gastric ulcer, the painful symptoms persist, or when the usual treatment of the stomach for disturbances of motility does not lead to improvement.

2. *Perigastritis with the Formation of Tumors.*—When the symptoms of gastric ulcer persist for months or years, a tumor becomes apparent in the left epigastric region. The growth of the tumor is gradual, and the mass is often adherent to the anterior abdominal wall. Vomiting is frequently a symptom. It may not be possible to exclude the alternative of malignancy until after a somewhat extended period of observation.

Diagnosis.—Roentgen fluoroscopy is of great value in the diagnosis of perigastritis. Exploratory laparotomy is an acknowledged and necessary measure for the recognition and cure of this condition. Adhesions from gastric ulcer are by no means uncommon; in the postmortem room about 45 per cent. of the cases of gastric ulcer show more or less adhesion to neighboring organs. Fenwick's table of 123 cases shows the pancreas and liver to be the organs most frequently involved in the adhesions. Adhesion to the pancreas frequently saves the patient from the danger of perforation. In cases of perigastric adhesions little or no loss of flesh is observed; the condition is seldom fatal. The paroxysmal character of the pain is supposed to be due to peristalsis, which

causes a dragging upon the adhesions. It is thought that many cases of "gastralgia," "hysteria," or "hypochondriasis," if carefully investigated, would be found to be due to intra-abdominal adhesions. Local tenderness is sometimes elicited, and more rarely still the matting together of the organs can be made out by palpation. Severe pain, in fact, is the most prominent symptom. It must be remembered, of course, that perigastric adhesions and an unhealed gastric ulcer may be associated. When the symptoms are due entirely to the adhesions, the pain is apt to be constant and of long duration, more pronounced when the stomach is empty than when it is full; it is not produced or increased by the taking of food. The situation of the adhesions will also influence the symptoms. For instance, if a band passes from the stomach to the colon, the contraction of either of these organs will cause severe pain; but if a large area of the stomach is fixed to the pancreas, it is not likely that the pain will be severe. The history of an old gastric ulcer is of the greatest value.

In regard to adhesions and perigastritis we are, unfortunately, able to make a diagnosis in only a very small percentage of cases. Perigastritis, unless there is a distinct disturbance of motility, is rarely a sufficient reason for surgical intervention. When firm immovable tumors can be palpated in the epigastrium, and carcinoma can be excluded, the existence of adhesions or epigastric hernia may be suspected. Adhesions may or may not interfere with the motility of the stomach. Those not interfering may be wisely left undisturbed, for we all know that severed adhesions are likely to re-form. In rare cases there may be an adhesion near the pylorus, predisposing to dilatation, that leads us to believe we have a case of organic obstruction of the pylorus. Morris has called our attention to adhesions around the liver, which he calls "cobwebs," that may cause many symptoms of indigestion. When these adhesions occur around the stomach, interfering with motility, they may produce symptoms suggesting dilatation. The methods of examination mentioned in Chapter XXIV on Motor Insufficiency will help us in the diagnosis.

Treatment.—Prophylaxis consists in the early diagnosis of gastric ulcer and its early cure, for the sooner an ulcer heals the less opportunity is there for the formation of adhesions. Of therapeutic agents, only fibrolysin and thiosinamin are worthy of consideration. These drugs may be used in the less severe forms of adhesions, cicatricial stenosis, and the so-called "hour-glass contraction." The treatment of cicatricial stenosis by fibrolysin has been described (see page 484). The so-called hour-glass stomach is to be treated in other respects as motor insufficiency of the second degree (see Chapter XXIV).

When a diagnosis of perigastritis has been made with reasonable certainty, too much time should not be spent with internal medi-

cation, inasmuch as surgical intervention is indicated. In simple adhesions good results have been obtained by simply breaking them up. When the condition is complicated with motor disturbance, a gastroenterostomy should be performed. The perigastric tumor must be treated surgically. It is important for the surgeon to bear in mind that there may be two ulcers and therefore two sets of adhesions in the same case. When the adhesions cannot be separated it may become necessary to perform pyloroplasty or gastrojejunostomy.

Promotion of visceral movement is the most efficient means of preventing adhesion of raw peritoneal surfaces—movement in bed, general massage, and mild laxatives.

CHAPTER XXVIII.

ARTERIOSCLEROSIS; SYPHILIS; TUBERCULOSIS.

ARTERIOSCLEROSIS.

ARTERIOSCLEROSIS consists of a thickening of the intima as a result of primary changes in the media and adventitia. The sclerotic condition may be diffuse or circumscribed; later in the progress of the disease it involves the media and adventitia.

Sclerosis of the abdominal arteries may be responsible for any one of the three following pathologic manifestations:

1. *Gastric Hemorrhages*.—The cause of the hemorrhages is miliary aneurysm of the gastric arterioles, developing on sclerotic bases. The diagnosis can be made with a reasonable degree of probability only in patients of advanced age who are affected with a general arteriosclerosis. The treatment of this form of hemorrhage is the same as that of gastrorrhagia from other causes.

2. *Gastric Ulcer*.—This condition is likely to supervene in vascular areas in which the blood supply has become defective in consequence of sclerotic obliteration of the arterioles. The treatment is that of the round or peptic ulcer.

3. *Abdominal Angina*.—Pain of a severe and paroxysmal nature sometimes follows sclerosis of the abdominal aorta and its branches. The attacks are apt to take place at night after bodily exertion or mental excitement.

Etiology.—Among the important factors producing sclerotic changes in the arteries are:

1. *Old Age*. Arteriosclerosis is preëminently a disease of the later years of life, when it occurs as an involution process, an expression of the natural wear and tear to which the arteries are subjected. Longevity is largely a vascular question; the relationship is well expressed in the adage, "A man is as old as his arteries."

2. *Toxic Factors*.—Alcohol, lead poisoning and gout are important factors in the causation of arteriosclerosis.

3. *Syphilis*.—Syphilis, inherited or acquired, is a most important cause of sclerotic changes in the arteries of the young and the middle-aged.

4. *Overeating*.—Overeating is an important etiologic factor.

5. *Overwork*. Muscular overwork or prolonged and severe exercise tends to produce hypertension of the arteries by increasing the peripheral resistance.

6. *Toxemia*.—As to the poisons generated by the ordinary bacteria of the intestine, Metchnikoff believes that his experiments have now established beyond question that small doses of paracresol and indol, acting on the organism over a longer or shorter period, are capable of inducing chronic lesions of a sclerotic nature. Such lesions are the very ones that are most frequently encountered in senility. His latest experimental and chemical research demonstrates further that the phenols and indol found in the stool and urine are not the excreta of our tissues, but the products of permanent microbial flora. It is not unreasonable to assume that the digestive tract can constantly harbor an injurious flora, the source of chronic poisoning, leading to arteriosclerosis. (See Chapter XXXIX.)

Pathology.—The changes to be described under this heading are of a degenerative character, and have an important bearing upon the integrity of the arterial walls as well as upon the viscera supplied by the sclerosed arteries.

Owing to the proliferation of endothelium and to an increase in the connective tissue of the intermediate layer, a thickening of the intima results, which may wholly or practically occlude the lumina of small arteries. In the large arteries the new tissue may form beneath the endothelium diffusely or in circumscribed masses. The endothelium may remain intact or it may undergo various changes; it may proliferate, or it may become fatty or necrotic. The newly formed fibrous tissue of the intima is apt to undergo fatty degeneration, to become necrotic and to disintegrate. Cavities of varying size, containing disintegrated tissue, fat and cholesterol crystals, develop in the newly formed tissue into what have been designated atheromatous cysts. These cysts may extend toward the lumen of the vessel, opening into which they may give rise to emboli or form rough ulcers (often with undermined edges) upon which thrombi may form. In the newly formed tissue of the intima, as well as in the necrotic foci, and in the detritus of the cysts, calcification may occur. Fatty degeneration, atrophy and calcification may occur in the muscularis and adventitia of the involved vessels.

Symptoms.—These general arteriosclerotic changes give rise to symptoms which are attributed by the patient to the stomach and some primary disorder of digestion. The patient can never be fully convinced but that if his stomach were in good condition he would be well again. Among the subjective symptoms are a feeling of fulness in the epigastrium, pain under the ensiform cartilage and down the left arm, gaseous eructations, and extreme nervousness and anxiety. There is, as a rule, immediate relief on belching. These are the leading symptoms which were complained of by any number of patients, and which subsided after appropriate treatment directed to the vascular system. The

digestive disturbances are secondary to primary changes in the arterial system. In fact, not only the stomach but the whole intestinal tract is affected by the changes. Sclerosis of the intestinal bloodvessels in many cases does not produce any subjective or objective symptoms. Sometimes, however, mild or severe disturbances of the bowels may be excited by an extensive sclerosis of the abdominal vascular apparatus. Obstinate flatulence and derangement of absorption may often be found to be due to circulatory disturbances in the abdominal region in consequence of sclerosis of the arteries. The violent and excruciatingly painful attacks of *angina abdominalis* develop as a result of arteriosclerosis, just as *angina pectoris* is induced by sclerosis of the coronary arteries. The occlusion, by sclerosis, of certain branches of the mesenteric artery may give rise to grave abdominal disturbance. This may be the cause of intestinal ulcers, necroses, and severe hemorrhages. Only within recent years has it been recognized that sclerosis of the abdominal arteries may cause distressing abdominal symptoms, an abdominal analogue to *angina pectoris*. The syndrome has been called by various writers abdominal apoplexy, intra-abdominal intermittent claudication, intestinal miopragia, and intermittent ischemic dysperistalsis. The intestinal crises may coincide or alternate with similar disturbances in the stomach from similar arteriosclerotic lesions.

Diagnosis. In his diagnosis the physician should not be led astray by the complaints of his patient as to indigestion, pain in the stomach, distention and belching, but should make a careful search for the underlying cause of the digestive disturbances.

In patients past middle life who complain of pain in the stomach, distention after eating if they attempt any physical exertion, and dyspnea, relieved by belching of gas—especially when nocturnal seizures, accompanied by distention, heart disturbances, dyspnea, and great anxiety, are prominent symptoms—a careful examination of the vascular system will, as a rule, reveal the real cause of the condition. Such examination usually shows a heart somewhat enlarged, an aortic second sound sharp and snapping, a murmur over the aortic area and rough sounds over the aorta itself, pulsation in the episternal notch, attacks of pain over the precordial region radiating to the arm, marked tenderness over the abdominal aorta down to the navel, urine perhaps increased in amount or containing albumin in small quantity, or both increased and albuminous. An important sign is the high blood-pressure, which is always suggestive. All these point unmistakably to the circulatory system as the real cause of the trouble.

Treatment.—The treatment primarily must be directed toward the general arteriosclerosis. Improvement is sought by various measures that are well known, such as lactovegetable diet, hydrotherapy, balneotherapy, gymnastics, electricity, and medication.

The diet should be plain, nutritious, and easily digestible. The evening meal should be limited in size, to minimize the formation of gas. Alcohol, tobacco, tea and coffee should be interdicted in arteriosclerosis, or restricted to a minimum. Moderation in eating and drinking is essential to the arrest of the pathologic process going on in the arteries. Animal foods should be restricted, for the digestion of these foods develops substances that add to the abnormal conditions already prevailing in the body (see page 422).

Bathing, fresh air, moderate exercise, and attention to the bowels should enter into the hygienic treatment. Intense excitement should be avoided. Many patients require absolute physical and mental rest, especially as they enter the stage of myocardial incapacity. By proper clothing the peripheral circulation should be protected from sudden changes of temperature.

Of medicinal agents for the treatment of arteriosclerosis producing gastro-intestinal symptoms, sodiosalicylate of theobromin in doses of 0.5 to 1 Gm. (8 to 15 grains) three times a day is recommended. I have found this drug so satisfactory in bringing about an amelioration of symptoms that I have used it as an aid to diagnosis in doubtful cases. Its effects depend on its powerful action in overcoming the vascular spasm and dilating the arterioles so that they allow a greater flow of blood to the sclerosed areas. It has been suggested that sodiosalicylate of theobromin may neutralize the effect of some toxic agent which tends to irritate the vasomotor centers and cause contraction. Whatever the exact mode of action, its effects are very satisfactory, and its use may be continued for one or two weeks or even longer without harm. The effect may then be maintained by the use of tincture of strophanthus, 5 to 8 drops three times a day. Strophanthus has been observed to act so much like sodiosalicylate of theobromin that it is used in place of the latter in some cases in which expense is a great consideration. Erythrol tetranitrate lowers blood-pressure and maintains its vasodilator effect for a longer period of time than other preparations of the same class. Its influence upon the blood-vessels is manifest in fifteen to twenty minutes after the dose is administered and persists for three or four hours. The dose is 0.02 to 0.06 Gm. ($\frac{1}{2}$ to 1 grain). Variation in the amount and frequency of the dose is regulated by the demands of the case and the effect on the patient. The nitrites act on the preganglionic endings of the sympathetic nerve fibers, which are the inhibitory nerves of the intestine.

Papaverin hydrochlorid is a powerful dilator of the coronary artery and lowers general blood-pressure by directly dilating the bloodvessels, especially the splanchnic and peripheral vessels. In doses of 0.03 Gm. ($\frac{1}{2}$ grain) it relaxes muscles of the cardia, pyloric sphincter, and intestine. Benzyl benzoate has been used with great benefit in abdominal angina. It is a powerful vasodilator with no

depressing effect upon the heart. Its action is due to the inhibitory and tonus-lowering or spasm-relaxing action of the benzyl radical on smooth muscle (see page 276).

In prescribing the iodids in arteriosclerosis, the continued good effects of this medication are to be obtained only by gradually increasing the dosage until the sluggish live cells are sufficiently stimulated and enough degenerated cells destroyed to insure the restitution of function in the tissues. The prolonged administration of small doses fails to accomplish permanently favorable results; but large and progressively increasing doses produce strikingly good results, in early cases particularly. In the use of potassium iodid the patient is to be started with a 0.6-Gm. (10-grain) dose three times daily, which should be daily increased until 4 to 4.6 Gm. (60 to 70 grains) is given each day. Iodism can be prevented by the careful exclusion of acids from the diet, and by neutralization of the acid contents of the stomach by means of any agreeable alkali. In this connection it should be stated that potassium iodid reduces the viscosity of the blood and in that way assists in the relief of blood-pressure and in invigoration of the arterial tissues.

Thyroid extract has been administered in arteriosclerosis, with favorable results, due to its power to control high arterial pressure.

In the endeavor to combat arteriosclerosis by promoting vascular metabolism, strengthening the vasomotor nerves, and reducing the tension of the vessels, Trunczek resorted to the hypodermic administration of the inorganic blood salts. His results have been confirmed by Tessier, Levy, Merlken, Zanoni and others; the serum acts on the calcium phosphate, relieves dyspnea by increasing the alkalinity of the blood, has a direct effect on the heart and the vascular endothelium, and stimulates the vasomotor system. Levy has found that, given by mouth, the salts have the same effect as when administered hypodermically. Under the trade name antisclerosin a combination consisting of these blood salts is available. Each dose (two tablets) contains:

	Gm. or Cc.	
R—Sodii chloridi	0 8	gr xij
Sodii sulphatis	0 08	gr i½
Magnesi phosphatis,		
Sodii carbonatis exsiccati	AA 0 03	gr ss
Sodii phosphatis,		
Calci glycerophosphatis	AA 0 025	gr. ½

This represents at least 15 Cc. of Trunczek's serum, and equals the saline contents of about 150 Cc. of blood serum.

Though certain cases are incurable, antisclerosin will often relieve the subjective and objective symptoms in even severe cases. Its chief field of usefulness is as a prophylactic.

SYPHILIS.

The occurrence of syphilis in the stomach is not as infrequent as we have been led to believe. Statements from various sources tend to show that about one out of seventy-five of our gastric cases is syphilitic. All the cases so far observed have occurred during the tertiary stage of the disease. Gastric syphilis appears in three forms—(1) specific ulcers of the stomach; (2) specific tumors; (3) specific stenosis of the pylorus. The syphilitic ulcer is the most frequent manifestation of syphilis affecting the stomach; it may develop as a result of disturbances in the circulation affecting circumscribed areas of the gastric mucous membrane, and having as its primary cause a specific endarteritis. Ulcers may also arise from the disintegration of gummata in the submucous coat of the stomach. The gummatous ulcer develops in the submucosa, while that caused by specific endarteritis is essentially an ulcer of the mucosa; neither differs in any way from the ordinary round ulcer, except that the specific ulcer is much less responsive to treatment. In syphilitic ulcer we find subacidity, rather than hyperacidity as in round ulcer. In specific stenosis of the pylorus the whole stomach may become cirrhotic, due to an increase of connective tissue. Many cases of that rare disease known as *linitis plastica* (gastric cirrhosis) have been supposed to be caused by syphilis, but we now know they are caused by carcinoma (see page 546).

Diagnosis.—It is a very difficult matter to make a diagnosis of syphilitic ulcer, and the clinician must rely upon the known presence of syphilitic infection in order to be at all certain that the gastric ulcer is of luetic origin. The presence or absence of the *Treponema pallidum* (*Spirochæta pallida*) is not to be depended upon in determining the presence or absence of ulcer of syphilitic origin. These microorganisms are often absent in cases of undoubted syphilis; on the other hand, Koch, using the Levaditi stain, found, in cases of undoubted carcinoma of the lung, organisms of the typical appearance of the *Treponema pallidum*. A positive Wassermann reaction materially assists in the diagnosis. The luetin test of Noguchi, involving a cutaneous reaction after injecting into the superficial layers of the skin dead cultures of *Treponema pallidum* is usually positive in tertiary syphilis; but the test cannot be depended upon in the primary and secondary stages. Under specific treatment during the secondary stage, especially after the administration of arsphenamine, a positive luetin reaction can be obtained. The test is valuable in obscure tertiary manifestations, where the Wassermann reaction often fails. The disappearance of the luetin reaction, according to Noguchi, may mean that the patient has fully recovered from the disease. Syphilitic ulceration of the stomach should be distinguished clinically from carcinoma and the gastric crises of locomotor ataxia.

The differentiation between syphilitic ulcer and other conditions producing dyspeptic symptoms may be further made by a course of antisyphilitic remedies, such as potassium iodid, which will usually ameliorate syphilitic symptoms while the same treatment would have the reverse effect upon ordinary cases of gastritis.

Garel's diagnostic sign is prolonged and painful dysphagia. There may be no other symptom suggesting the possibility of syphilis. In all cases of prolonged dysphagia referred to the pharynx, a Wassermann test should be made.

Treatment.—The treatment of syphilis of the stomach consists in the methods and agents employed in cases of gastric ulcer and gastric hemorrhage, together with such specific remedies as arsphenamine, mercury, and the iodids, the latter being tolerated well by the luetic stomach. (See Chapters XXV and XXVI.)

Arsphenamine (salvarsan) is a yellowish crystalline arsenical compound, not unlike iodoform in appearance, and must be kept in hermetically sealed ampoules, for it becomes very toxic when exposed to the air. For administration it must be handled with the greatest care and every detail in the prescribed technic carried out. The dose (for intramuscular or intravenous injection) is 0.3 to 0.6 Gm. (5 to 10 grains).

Neoarsphenamine (neosalvarsan). Ehrlich has given us an improved salvarsan (neosalvarsan), now known as neoarsphenamine. It is a fine powder, resembling salvarsan, but somewhat more yellowish in color and turning reddish on exposure to the air. It dissolves readily in water, which becomes yellow without stirring. The solution is neutral in reaction, which eliminates the use of sodium hydroxid for alkalinizing purposes. Neoarsphenamine can be given both intramuscularly and intravenously and in larger doses than arsphenamine. On account of its non-irritating character it can be given with greater safety by simply dissolving it in water. Since it can be so easily manipulated, its value is obvious. The dose of neoarsphenamine is 0.9 Gm. (14 grains) for men and 0.75 Gm. (12 grains) for women.

Ehrlich has also introduced *sodium-salvarsan* (No. 1206A), which is supposed to combine the advantages of the two earlier preparations. It is a fine golden-yellow powder, readily soluble in water. It contains the same proportion of arsenic as arsphenamine and neoarsphenamine. The dose ranges from 0.6 to 1 Gm., without any discomfort from the larger doses. The drug has been found to be absolutely non-toxic, and the injections do not produce the slightest reaction. It can therefore be given in cases of syphilis associated with apoplexy, diabetes, nephritis or other diseases. Sodium-salvarsan is really old salvarsan so modified as to be as easily given as neoarsphenamine (neoarsphenamine).

A large number of patients have been successfully treated with these preparations which have been used in all stages of syphilis,

from the primary chancre to the deep specific lesions of the central nervous system. They possess apparently the peculiar property of killing and exterminating the *Treponema pallidum* (*Spirochaeta pallida*), wherever it is to be found in the body. They are known to cure syphilis in all three stages, particularly when combined with a course of mercury. The first effect is manifest in modification of the appearance of syphilides, mucous patches, gummata, and chancre. The shooting pains, the girdle sensation, and the tabetic crises are relieved almost immediately. But a single dose of arsphenamine will not eradicate the disease. In the initial stage of the infection, arsphenamine is more effective than mercury; and when mercury and the iodids have failed, arsphenamine is often beneficial. From the observations of many clinicians, however, it may be fairly concluded that the best results are obtained by giving mercury as well as arsphenamine.

The intravenous administration of bichlorid of mercury, in doses of 0.02 Gm. ($\frac{1}{2}$ grain) dissolved in 10 Cc. ($2\frac{1}{2}$ drams) of freshly distilled water, is of great benefit. To avoid as far as possible the danger of causing phlebitis by placing mercury in the vein, the injection should be performed very slowly, and a new vein chosen for each consecutive injection. The mercury will be much less apt to injure the vein if a syringe with a capacity of about 20 Cc. be used, so that the mercurial solution in the syringe may be diluted with the patient's blood before being injected into the vein. After the needle has entered the vein, by traction on the piston the physician should withdraw sufficient blood to fill the barrel of the syringe, when, without removing the needle, the mercurialized blood should be slowly introduced. The injections may be given at intervals of four to six days and continued until the Wassermann reaction is negative.

Syphilitic stenosis of the pylorus is an exceedingly rare condition, which may be due to cicatrices of syphilitic ulcers or tumors and gummatous infiltration in the region of the pylorus. The treatment for this condition is the same as for motor disturbances (see Chapter XXIV).

General Treatment.—Patients suffering from syphilitic disease of the stomach should be kept at rest, preferably in bed. The food should be of a simple, unirritating kind, its precise character depending upon the severity of the symptoms. In very severe cases it may be necessary to resort to rectal alimentation. In any case it is advisable to inaugurate the dietetic treatment by the use of milk. As the condition of the patient improves, the quantity and variety of the food may be slowly increased, and jellies flavored with lemon and sweetened, junket, eggs lightly cooked or beaten up with milk, custards, or tapioca pudding may be permitted. This regimen may be followed later by bread and

butter, fish, chicken, rabbit, or veal. No meat should be allowed for at least six months.

The bowels should be carefully regulated, and for the control of constipation resort should be had to saline cathartics.

TUBERCULOSIS.

Gastric tuberculosis is a very rare condition, and when present is usually secondary to or associated with tuberculosis of other organs.

Forms.—Three forms of tuberculosis of the stomach have been recognized, namely: (1) Miliary tuberculosis—always secondary to general miliary tuberculosis; it cannot be diagnosticated, and is therefore not subject to treatment. (2) Tuberculous ulcer. This occasionally produces disturbances similar to those produced by peptic ulcer; hemorrhages are not infrequent, and perforation is more likely to occur than with peptic ulcer. Tuberculous ulcer has been attributed to the swallowing of tuberculous sputum. It may also result from infection through the blood and lymph routes. The treatment is that of peptic ulcer. (3) Tuberculous granulation tumors—located on the pylorus or in the region of the pylorus. The symptoms resemble those of gastric carcinoma, and the treatment is the same as for the latter. In tuberculous stenosis of the pylorus, success has followed resection.

Treatment.—The treatment of tuberculosis of the stomach is the treatment of tuberculosis localized elsewhere in the body.

CHAPTER XXIX.

TUMORS OF THE STOMACH.

CARCINOMA; SARCOMA; FIBROMA; FIBROMYOMA; LIPOMA; ADENOMA;
PAPILLOMA; POLYPI; HERNIA EPIGASTRICA.

CARCINOMA.

GASTRIC carcinoma consists of a malignant new-growth or tumor made up principally of epithelial cells and developing in the epithelial tissue of the stomach.

Etiology.—*Incidence.*—Of all the viscera of the body, the stomach is most frequently the seat of carcinoma. Of the total number of carcinoma cases reported, from 40 to 45 per cent. are said to be carcinoma of the stomach. It occurs more frequently in males than in females. According to Wyss the death-rate from gastric carcinoma is nearly 2 per cent. of the total mortality; Bryant states that in New York City for the ten years immediately preceding 1896 it was 2.17 per cent. of the total mortality. In the United States census of 1912 we find 46,534 deaths from carcinoma; of these, 18,517 were of the stomach (39.75 per cent.). The frequency of this disease varies in different countries. There are certain regions in which it rarely occurs. Griesinger states that he has never seen a case of carcinoma of the stomach in Egypt, while Heinemann reports that he saw only one case in Vera Cruz during a period of six years. When we consider the advances that have been made in medical science in the way of refinement of diagnosis, it is evident that carcinoma is more frequently discovered and differentiated from non-malignant growths than was formerly the case. This of itself would account, in a measure, for the apparent increase in the prevalence of the disease.

In the development of carcinoma a certain individual predisposition is necessary, which may be either congenital or acquired, although we do not know exactly the nature of this predisposition.¹ Certain substances which would inhibit or defend the body against the formation of carcinoma may be absent. It has been shown that immunity to carcinoma bears a certain relation to the number of white lymph corpuscles present in the blood. The number of these may be increased in an active manner by the body itself in resisting the development of the carcinoma, or passively stimulated by the injection of lymph tissues. There may be a local initia-

¹ Caenoy, *Münchener medizinische Wochenschrift*, April 1, 1913

tion of carcinoma, such as trauma, injury, inflammation, a scar, or some congenital malformation. Or, possibly, there is a carcinoma organism, belonging to the group of hitherto unknown ultramicroscopic organisms, and which is brought to the human subject by an intermediate carrier. Whether this carrier is the bed-bug or other blood-sucking parasite, or one of the family of the small pin-worms carried by cockroaches, is not certain. Observations seem to point to a certain infection, not direct, but indirect through parasites. In all infectious diseases the virulence of the exciting agent is of great importance, for the healthy body has a certain power of resistance toward pathogenic agents in general.

Among predisposing if not strictly etiologic factors, gastric ulcer, the cicatrix of an ulcer, and chronic gastritis may be mentioned. Some surgeons attribute all gastric carcinomata to a previous ulcer of the stomach. In a study by Friedenwald¹ of 1000 cases of carcinoma in which careful records had been kept, there was a history of some previous digestive trouble in 232 cases (23 per cent.). Of the 232 cases 109 had had slight attacks of indigestion for a period of five years. Of the remaining 123 cases, 32 had had chronic indigestion more or less all their lives, mainly (in 29 of the 32) during the last five years preceding the present illness. Seventy-three cases gave a definite history of former gastric ulcer. It is therefore evident that of the 1000 cases but 23 per cent. presented a history of any previous digestive disturbance whatever, even in the slightest degree, and that but 7.3 per cent. gave a direct history of ulcer. If, therefore, all of the former digestive disturbances be considered as due to ulcer, the formation of gastric carcinoma from ulcer could not have taken place in more than 23 per cent.; if all of the cases with slight digestive disturbances be disregarded this percentage is reduced to 12.3 per cent.

From a study of 445 pathologic sections of gastric carcinoma, 339 of which were resected by the surgeon and 46 removed at necropsy, Wilson concludes that "practically all carcinomata develop on the site of a previous ulcerative lesion of the gastric mucosa."² This report is not in accord with our clinical experience. Albert Kucher³ states that he has personally examined the Mayos' specimens, and is convinced that much of what they labeled cancerous degeneration of ulcers was in reality merely atypical proliferation of epithelium.

Age.—Regarding the age at which gastric carcinoma occurs most frequently, Brinton has collected some interesting data. In

¹ American Journal of the Medical Sciences, November, 1914, p. 666.

² Wilson and McDowell, American Journal of the Medical Sciences, December, 1914.

³ Chronic and Duodenal Ulcer, Correspondenz-Blatt für Schweizer Aerzte, Basel, May 10, 1919, No. 19.

600 cases the year of death averaged fifty-two-sevenths of these between fifty and sixty. This writer places the maximum liability between sixty and seventy. Under twenty, the whole risk is less than one-fiftieth of what it is between twenty and thirty. Lebert gives us the following figures as to the age at which carcinoma of the stomach occurs: Under thirty years, 1 per cent.; thirty to forty years, 17.6 per cent.; forty to sixty, 60.7 per cent.; sixty to seventy, 16.3 per cent.; above seventy, 4.4 per cent.

Heredity.—The influence of heredity as a predisposing cause of carcinoma of the stomach is still an open question. The occurrence of carcinoma in one or more of the off-spring of carcinomatous parents has been noted, but not with marked frequency. Carcinoma has been known to attack persons whose health up to the time of the attack had been remarkably good.

Pathology. Gastric carcinoma consists of atypical epithelial proliferation having its starting point in the glandular cells and the epithelial lining of the secretory ducts. It develops into a tumor of varying size, sometimes attaining such dimensions as to occlude the lumen of the stomach. Carcinoma occasionally consists of more or less flat granulations and excrescences. In many instances there is a tendency to superficial and sometimes to deep ulceration and necrosis, infiltrating the walls of the organ until it becomes a cavity enclosed by carcinomatous tissue. As this process involves the bloodvessels, hemorrhage occurs.

Gastric carcinoma forms metastases by way of the lymphatics, as a rule. In some instances, especially in the medullary and scirrhous types, this occurs early. The glands draining the gastric area rapidly become involved, and metastatic nodules appear in the omentum and peritoneum, which in some cases is so massively infiltrated that dissection is difficult. Not infrequently so-called "precocious metastases" appear in the skin, in the bones, and in other distant organs. In very many instances growth of the tumor takes place by infiltration so that wide areas of the stomach wall are affected and become markedly thickened and inelastic. In the progress of such growth bloodvessels may be involved, and cells of the tumor, set free in the blood, are then disseminated to the liver, lungs, heart, and rectum.

Carcinomata differ widely not only in gross and microscopic structure but also in respect to rapidity of growth and degree of malignancy. Some grow with alarming rapidity (carcinoma simplex), while others remain dormant or nearly stationary for long periods of time (many adenocarcinomata, and the diffuse fibro-carcinomata).

Gastric carcinoma is most frequently located in the pyloric region (60 per cent.), and in such cases is prone to bring about a stenosis of the orifice and an accompanying dilatation of the stomach (see Chapter XXIV). Forty per cent. of carcinomata appear to

be equally divided between the lesser curvature (including the cardia) and the greater curvature. Diffuse cancerous infiltration of the stomach occurs in about 6 per cent. of the total number of cases.

Forms.—The forms of gastric carcinoma, in the order of their frequency, are: Medullary carcinoma, adenocarcinoma, colloid carcinoma, and scirrhus carcinoma.

Medullary Carcinoma (Including Carcinoma Simplex).—Medullary carcinomata are of two gross forms: one consisting of soft, rounded, large but circumscribed tumors, appearing chiefly in the cardia and fundus; the other a diffuse growth, appearing most frequently near the pylorus. The former is the "most aggressive and rapidly progressive form of gastric carcinoma" (Ewing). The latter is a type with which perforation is most frequent. Both tend to early metastases, early ulcerations, and hemorrhage.

Adenocarcinoma.—The adenocarcinomata of the stomach are relatively benign. For the most part they form circumscribed polypoid or fungating, and eventually ulcerating, masses. They appear near the pylorus, in which case ulceration takes place earlier, or on the curvatures, where they reach a larger size. Occasionally they seem to be the result of carcinomatous transformation of adenomatous polyps, and in some such instances there are no prominent gastric symptoms and no invasion of nodes.

Gelatinous or Colloid Carcinoma. The gelatinous or colloid carcinomata result from colloid or mucoid transformation of the cells of malignant growths. If it be that this change appears in medullary cancers, then other changes also occur, for in the colloid type invasion of the lymph nodes is later, growth by extension more frequent, and diffuse infiltration more marked than in the medullary form. The peritoneum is very frequently and extensively involved.

Scirrhus Carcinoma. It is the relatively large amount of fibrous tissue as compared with the epithelial tissue which gives the firmness to the growths called *scirrhus*. These are very firm, sometimes almost cartilaginous in appearance and texture. In the somewhat pearly substance may be seen small points or lines of a yellowish or reddish hue, which represent the epithelial tissue that is undergoing fatty metamorphosis. Such growths are slowly progressive. Ulceration is late, as a rule. Because of the character of the growth and the frequency of its localization at or near the pylorus, pyloric stenosis is a common result. But while it is ordinarily located in the pyloric region, it may involve a large portion of the stomach or even the whole organ (sclerosing pyloric and diffuse fibrocarcinoma; *limitis plastica*; see page 546). While ulceration occurs late, superficial erosions of large extent may appear early. The lymph nodes are involved regularly and early, and the metastatic nodules are prone to become large and are always more cellular than the original growth.

Ulcerocarcinoma.—Because of the frequency with which malignant epithelial changes occur in connection with chronic gastric ulcers, and because of the rather typical clinical history that is associated with such tumors, it is the custom of some writers to use the term "ulcerocarcinoma" in describing these cases.

Complications.—The portion of the gastric mucous membrane that is not directly involved in the carcinomatous process may function in a perfectly normal manner, especially during the initial stage of the disease. This, however, does not continue for any great length of time, owing to the progressive atrophic changes which take place in the gastric mucous membrane. In cases of gastric carcinoma complicated with pyloric stenosis and stagnation, a frequent feature is chronic gastritis with marked secretion of mucus and a decrease in the secretion of gastric juice.

Complications of gastric carcinoma may consist of adhesions to or rupture into neighboring organs, such as the intestine, gall bladder, pancreas, and liver. Perforation into the peritoneal cavity is rarely met. Other rare complications are rupture into the pleural cavity, subphrenic abscess, pyopneumothorax, and free bleeding from the carcinoma itself (gastric hemorrhage).

Symptoms.—The earliest symptoms of carcinoma of the stomach are pressure and fulness after eating. This mild sensation is superseded sooner or later by pain of varying intensity; the pain may be felt in the region of the stomach, directly in front or to the right or left of the median line, or it may be felt in the dorsal region. Pain is not a constant symptom, however; it is often absent, particularly when the carcinoma is situated on the lesser curvature. The patient is frequently annoyed by eructations due to decomposition of the food mass within the stomach. Anorexia is among the early symptoms, the patients manifesting a distaste for meat. In a few cases the normal appetite has been known to continue for a long time after the appearance of the initial symptoms; and in rare cases there is a markedly increased appetite. As a rule the patients complain of weakness and are disinclined or unable to work. They lose flesh rapidly. These symptoms progress until nausea and vomiting become troublesome features. Vomiting depends largely upon the location of the neoplasm; carcinoma of the pylorus is nearly always accompanied by vomiting, owing to stenosis of the pyloric exit. In carcinoma of the greater or lesser curvature there may be no vomiting at any time throughout the course of the disease. When vomiting becomes severe, great thirst is experienced, and a marked diminution in the quantity of urine excreted is noted.

The inability to assimilate food results in rapid loss of weight. This condition becomes so marked that many patients die of inanition.

Statistics show that 60 per cent. of the carcinomata of the stomach

attack the pyloric region, 20 per cent. the lesser curvature and cardia, and 20 per cent. the greater curvature. Carcinoma of the pylorus seems to be associated with hypersecretion, while carcinoma of the lesser curvature is more often associated with achylia.

In carcinoma of the cardia the subjective symptoms are most insidious. Difficulty in deglutition is one of the first distinct signs confirmatory of the diagnosis; it results from occlusion of the lower end of the esophagus. The patient finds that he cannot swallow solid foods with ease; he experiences a sensation of the food "sticking fast" before it enters the stomach. Pain results from the movement of the food through the stenotic cardiac orifice.

As the disease progresses the patients acquire a characteristic cachectic appearance—loss of flesh and sallow complexion. In the later stages of the disease anemia supervenes; the percentage of hemoglobin and the number of red blood-cells are much below normal. The anemic condition may be due to hemorrhages, to insufficient nutrition, or to the effect of toxins from the carcinoma. Hemolytic substances have been discovered in the gastric contents of carcinomatous patients. Not infrequently there is edema in the region of the ankles.

Diagnosis. Examination of the stomach in a typical case of gastric carcinoma reveals a tumor of varying location, size and shape. Tumors of the pylorus are usually located to the right of the median line; with the stomach in the normal position it is impossible to palpate such tumors, owing to overlapping by the liver; when not concealed by the liver they may be felt below the right border of the ribs, protruding during deep inspiration. When the stomach has descended it may be palpated at varying distances below the ribs. Tumors of the lesser curvature may likewise be so covered by the liver as to render palpation impossible. The usual site of tumors of the greater curvature and of the fundus is at the level of the umbilicus or below it. These differ from neoplasms of the pylorus in the absence of symptoms of obstruction produced by pyloric stenosis. When the neoplasms have formed adhesions to neighboring organs of the abdominal cavity, they are found to be immovable or only slightly movable on palpation. Motor insufficiency supervenes in cases of carcinoma of the pylorus in proportion to the degree of stenosis present. The most frequent seat of metastasis is the liver, which, when affected, usually shows marked enlargement. When the carcinomatous growth has progressed for a considerable length of time the supraclavicular glands of the left side may be enlarged.

Carcinomata are found, however, to vary in a marked degree from the type here outlined. The latent stage of carcinoma is often prolonged. The tumor may not be discovered; subacidity or achylia may be diagnosed from an examination of the gas-

tric contents, with no further objective symptoms which would point to the presence of malignant growth. Differing from our typical case, free hydrochloric acid may be present for a long time after the initiation of the carcinomatous process, while the appearance of lactic acid may be long delayed or never present. In such cases it is extremely difficult and often impossible to make a diagnosis with certainty. Exploratory laparotomy is often justifiable.

Carcinoma of the cardia, as a rule, offers resistance more or less marked to the passage of a sound; especially is this the case when the lower portion of the esophagus is involved. This condition can be definitely diagnosed by means of the Roentgen ray (see Plate XI, Fig. 2). Roentgenology is frequently of great assistance in the diagnosis of gastric carcinoma (see Chapter V—Plate XIV, Fig. 1; Plate XV, Figs. 3 and 4; Plate XVI, Fig. 1).

An examination of the stomach contents in typical cases of gastric carcinoma reveals the presence of lactic acid (see page 97) and the bacilli of Boas-Oppler (Fig. 10, *B*); such cases are marked by the absence of free hydrochloric acid. The color of the gastric contents is frequently brown or of a coffee-ground appearance, owing to hemorrhages from the carcinomatous growth. Microscopically, blood and pus are both seen in the gastric contents in cases of ulcerated carcinoma. The test-diet stool is characteristic (see page 123). Occult hemorrhages are demonstrable in over 90 per cent. of the cases by examination of the feces. In cases of achylia, pepsin is often found in the urine (see page 97).

For the glycytryptophan, Salomon, Wolff-Jungmans and Gluzinski tests, see pages 87 and 88.

Serologic Reactions.—While serologic reactions are inconclusive, and even the most suggestive are still in a definitely experimental stage, much progress has been made that renders it necessary to review the most important of these reactions.

Hemolytic Reactions.—In the growth and breaking down of malignant tumors, as has been found by clinical investigation, substances are formed and set free that dissolve the red blood-cells. To these substances, or lysins, the anemia and cachexia of malignant disease have been ascribed. Elsberg, Neuhof, and Geist, reasoning on the assumption that the blood serum of patients suffering from malignant disease contains hemolysins, while that of normal individuals or those suffering from other diseases does not, maintain that in this blood phenomena we have possibly a valuable agency for the diagnosis of malignant disease. They have accordingly made use of the hemolytic property of the blood serum of carcinomatous patients. They inject subcutaneously into the forearm of the suspected carcinomatous subject 5 minims of a suspension of washed human blood corpuscles (1:5) in salt solution. The hemolysins in the blood serum of a carcinomatous subject attack the corpuscles so that a reaction shows in two to eight hours. The skin at the site of injec-

tion exhibits the effect in a color varying from brownish-red to bluish. The reaction has been found to be positive in 89.9 per cent. of the carcinomatous cases in which the test has been made.

Kelling has shown that normal blood serum will hemolyze the red corpuscles of hen's blood, while the blood serum of carcinomatous patients will not. The test is made by adding 1 Cc. of a 5-per-cent. saline suspension of the hen's corpuscles to 0.1 Cc. of the patient's serum.

Antitryptic Reaction.—When the blood serum of a carcinomatous patient is allowed to remain on a Loeffler serum plate, it digests itself. On adding normal serum, digestion is instantly retarded, proving the existence of an antiferment in normal blood serum.

The Mustagmin Reaction.—This reaction depends upon the fact that the serum of a carcinomatous person, if treated with a standardized reagent containing an antigen extracted from carcinomatous tissue, will undergo decreased surface tension and the drops will become smaller. They must be measured accurately with an instrument of precision known as the stalagmometer of Traube. The serum of a healthy person will not undergo such change.

The reagent is obtained by cutting up the carcinomatous tissue and drying *in vacuo*; 0.5 Gm. of this powder is then extracted with 25 Cc. of methyl alcohol at 50° C. in a closed vessel for twenty-four hours, with occasional agitation. This alcoholic solution is then diluted with distilled water to such a point that when titrated with known carcinomatous serum it will produce an increase in the number of drops in a given volume to the extent of 50 per cent. as compared with normal serum. To 9 Cc. of the serum to be tested, diluted to 180 Cc. with saline solution (0.85-per-cent. sodium chlorid), 1 Cc. of the antigen emulsion, standardized as above, is added. After incubation for one hour at 50° C. the number of drops in a given quantity are counted. The control consists in adding distilled water instead of antigen emulsion to the diluted serum and incubating under the same conditions—the number of drops in a given quantity being counted both before and after.

Abderhalden Reaction.—The Abderhalden serum reaction depends upon the fact that there is present in the blood of patients suffering from carcinoma a peculiar digestive ferment not found in the blood of healthy persons. This ferment is produced by the organism in an effort to protect itself against the growth of the carcinoma, hence it is called "protective." It is capable at times of completely digesting the carcinomatous cell invasion, thus curing the patient. When the invasion, however, has advanced as far as tumor formation, the body is unable to combat it.

The technic of the serum reaction is as follows:

The blood is obtained as for the Wassermann test, about 10 Cc.

being required. It must be chemically as well as bacteriologically clean. Care should be taken not to shake the tube containing the blood, as shaking is apt to dissolve some of the hemoglobin, and the presence of hemoglobin in the serum invalidates the test.

Carcinomatous tissue, to be used in the test, is obtained by cutting up pieces of carcinoma of the breast, thoroughly washing in distilled water until the biuret test applied is negative, then boiling for thirty minutes, after which the material is put into chloroform-water and kept on ice. A small portion of this carcinomatous tissue is placed in a dialyzing thimble, and 2 Cc. of the blood serum to be tested is added. The thimble containing the mixture is placed in a proper container filled with distilled water. Toluol is added to the dialyzer and the container to prevent putrefaction. The whole apparatus is placed in a thermostat and incubated for sixteen hours. At the end of this time the distilled water surrounding the thimble containing the serum and carcinomatous tissue is tested for the presence of peptone by the ninhydrin test. A positive reaction for peptone shows that the blood serum in the thimble contains a proteolytic ferment capable of disintegrating carcinoma. Ninhydrin reacts to aminoucid as well as to peptone and is preferable to biuret, which reacts only to peptone. Controls are run to show that the serum alone and carcinomatous tissue alone do not give positive reactions.

The test has been found correct in a number of cases and confirmed by operation or autopsy.

Blood-sugar Tolerance Test. Friedenwald and Grove¹ believe this test to be of distinctive value in distinguishing between carcinoma and other diseases of the digestive tract. The test is not specific of carcinoma, but it has an important bearing if diabetes, nephritis, tuberculosis and thyroid disturbances can be excluded. It has been known for some time that hyperglycemia is present in many carcinomatous cases, especially of the gastro-intestinal tract. In carcinoma of the stomach or intestine the blood-sugar tolerance test shows a characteristic curve which differs from that observed in carcinoma of other regions of the body. This curve presents a high sugar content, even in the fasting state, followed by an initial rise up to 0.24 per cent. or even higher within forty-five minutes after the ingestion of the dextrose, remaining at this level for at least two hours and at no time falling below 0.20 per cent.

The test is carried out in the following manner: After a night's fast, 100 grams of dextrose is dissolved in 300 Cc. of black coffee and given to the patient before breakfast. Just before this is given, blood is taken for a sugar test, and this procedure is repeated forty-five minutes and one hundred and twenty minutes after the ingestion of the sugar. In an individual in good health there is an increase

¹ Friedenwald and Grove: The Blood-sugar Tolerance Test as an Aid in the Diagnosis of Gastro-intestinal Cancer, American Journal of the Medical Sciences, September, 1920.

in the blood-sugar, reaching its maximum in forty-five minutes, and then gradually falling to normal, which is 0.08 to 0.12 per cent. In patients with carcinoma of the stomach or intestine the blood-sugar shows a rise in forty-five minutes, after which time it may continue to rise or remain stationarily high for two hours after the glucose meal. Subsequently the blood-sugar gradually recedes, becoming normal in three to four hours after the feeding.

Linitis plastica hypertrophica (leather-bottle stomach) is a sclerosing pyloric and diffuse fibrocarcinoma. It is a condition in which there is a great thickening of the walls of the stomach, due to fibrosis involving mainly the submucous and to some extent the other coats. The mucous membrane itself does not seem to be involved. The hypertrophy is so great that the lumen of the stomach is very much reduced. The fibrosis may be limited in extent, though usually the entire stomach is involved. The thickened wall is very rigid and may be six to ten times the normal size. The duodenum is rarely implicated. The connective-tissue proliferation is the most striking feature of this disease. The disease is one of adult life, and the patients are usually between the ages of forty and sixty, and about twice as often men as women. Roentgen-ray examination offers the best means of recognizing the lesion. The Roentgen fluoroscope shows the rapid emptying of the stomach as the bismuth flows without hindrance through the rigid gastric lumen into the duodenum. Serial roentgenograms indicate the degree of stenosis (see page 142).

Treatment.—The treatment of carcinoma of the stomach is essentially surgical. The physician should therefore endeavor to ascertain precisely the chances offered by operation in each individual case before employing internal medication, which is at best palliative and in no sense curative. As soon as the diagnosis of carcinoma of the stomach is confirmed, the case should be referred to the surgeon without delay. The value of surgery in early diagnosis is shown by my longest living case. She had one half of the stomach resected for pyloric carcinoma five years and eight months ago, and at this writing she seems perfectly well.

INTERNAL TREATMENT. Internal or palliative treatment is indicated in those cases which, after careful study and examination, are found to be unsuitable for surgical treatment. Medication must proceed along symptomatic lines. An endeavor should be made, on the one hand, to retard or inhibit the growth of the neoplasm, while on the other the subjective symptoms of the patient should be relieved to the greatest possible extent. Complete rest, both physical and mental, should be procured for the patient; he should occupy the recumbent position as much as possible, and retire early at night, since physical rest conserves the heat of the body; in this way the nutrition may be most advantageously maintained.

Diet.—Diet is of first importance in the internal treatment of carcinoma of the stomach. Of necessity small in quantity, it should be limited to articles of food with a high nutritive value; food containing the greatest number of calories, and which is at the same time non-irritating in its nature, should be prescribed.

The regimen for a carcinomatous patient should be of such variety as to keep the appetite stimulated as long as possible. Anorexia is found to be the greatest impediment to the nutrition of these patients. Owing to the tendency to stagnation of the food mass in the stomach, and consequent fermentation, the meals should be small and frequent. The character of the diet should be adapted as largely as possible to the condition of the gastric secretion—free hydrochloric acid being regarded as, to a certain extent, an indicator for the prescription of proteins. The reader is referred to Chapter XXIII on Subacidity and Anacidity for dietary measures covering those conditions of secretion. The question of motor disturbance should be kept in mind, and in the presence of marked stenosis the diet should be that laid down for the treatment of motor insufficiency of the second degree (see Chapter XXIV). In all cases of carcinoma of the stomach the diet should be of liquid or semiliquid consistency; the necessity for this is greater in the more marked stenoses of the pylorus and in cases with a tendency to hemorrhage. Of the liquid nutrients, milk occupies the first place; it may be prescribed very much according to the desires of the patient—alone or with tea, coffee, cocoa, or leguminous flours. Should milk become distasteful, buttermilk, sour milk, kefir, milk of almonds, milk soups, all form agreeable substitutes. Tastily prepared soups made from leguminous flours with eggs and butter, vegetable purées, flour puddings with fruit sauces, and malt extract free from fermentative processes, constitute valuable dietetic agents in this condition. Next to milk, eggs are most suitable for these patients; they may be prescribed soft-boiled, scrambled, as omelets, or raw beaten up with sugar and wine.

Fat may be prescribed in large quantities so long as the fermentative process in the stomach is not too pronounced; it should be in the form of butter, olive oil, or chocolates rich in fat. Meat should be given thoroughly boiled or roasted, and finely divided, preferably in the form of chopped meat. Should meat become distasteful, meat jellies may be tried; or, if the patient's repugnance to meat in any form is marked, it would be well to omit it altogether from the dietary for a few days. Light cheese may be prescribed. Zwieback, biscuits and toast must be softened before being eaten. All vegetables should be served in the form of purée.

The habits of life and the desires and tastes of the patient should not be disregarded entirely in prescribing diet. Various

kinds of delicacies may be incorporated in the menu. The mental impression produced by the addition of a few luxuries, as well as the preparation and serving of food in an attractive manner, is sure to have a favorable effect upon the patient. Monotony in diet should be carefully avoided, to keep up a fair appetite and counteract as far as possible the distaste for food which is too often a characteristic symptom of the disease. I would say, then, that a patient without marked stenosis of the pylorus may partake of a wide range of food so long as the various dietetic articles agree with him. It is often possible to keep the patient fairly well nourished for a considerable length of time. The greatest obstacle is encountered with that class of patients whose financial circumstances will not permit of such a varied diet as outlined.

In case of gastric hemorrhage resulting from carcinoma of the stomach, the patient should assume the recumbent position. The diet in this condition should be as prescribed in Chapter XXVI on Hemorrhage.

In cases in which it is extremely difficult to maintain nutrition by oral feeding, as in severe pyloric stenosis with troublesome vomiting, nutrient enemata should be employed, but only as an adjunct to oral feeding. Exclusive rectal alimentation should not be attempted, even for a few days, since it has been found that in carcinoma inanition results more rapidly under this regimen than with even a minor degree of oral alimentation alone (see page 243).

Treatment by Lavage. Washing out the stomach in gastric carcinoma is an important auxiliary to the dietetic treatment. Lavage is indicated when the motor function of the stomach is disturbed. It is especially indicated in pyloric stenosis with motor insufficiency of the second degree, as well as in motor insufficiency of the first degree. In the latter condition it is not necessary to wash the stomach every day. In motor insufficiency of the second degree, however, daily lavage should be performed, preferably at night before dinner. This daily lavage has the happy effect of relieving patients of many of their distressing symptoms; vomiting ceases, the pains decrease in severity, the appetite improves, and there is a marked improvement in the nutrition. Patients take on new hope, which is an important matter in the treatment of gastric carcinoma. Lavage, however, will not arrest the cachexia resulting from carcinoma; yet, in spite of the gradually progressive weakness, patients remain free from many subjective symptoms which would otherwise render their existence a greater burden. Lavage should be followed up by irrigation with antifermentative solutions, especially when there is marked formation of gas as shown by eructations. The lavage process should not be prolonged, since it requires more or less effort on the part of the patient (see page 197).

Mineral-water Cures. Mineral waters have not been found satisfactory in the treatment of gastric carcinoma. Sojourn at the so-called health resorts has not been attended by any marked improvement in the condition of the patient.

PHYSICAL TREATMENT.—This consists of local applications in the form of moist trunk packings, or hot moist or dry stupes applied in the gastric region to counteract the feeling of gastric pressure, pains, and nausea (see Chapter XII). Massage and electricity are not indicated in gastric carcinoma.

MEDICINAL TREATMENT.—The use of drugs in the treatment of carcinoma of the stomach is confined almost entirely to the relief of distressing symptoms. Condurango bark has been employed most frequently as a medicinal agent for the stimulation of the appetite (see page 267), and was believed at one time to possess remedial virtues. But while no drug has been found to exert any influence upon the course of the disease, condurango is still worthy of trial as a stomachic. It ameliorates nausea, vomiting, and pain, and on this account is to be preferred to the other bitter tonics. It is administered in the form of a decoction:

	Gm. or Co.	
R—Corticis condurango	15 0	℥ss
Macerate for twelve hours with dis-		
tilled water	300 0	℥xij
Sig.—Tablespoonful three times a day, before meals.		

In addition to condurango bark, the cinchona preparations, tincture of gentian, and orexin may be prescribed (see page 267).

In subacidity and anacidity, pepsin and hydrochloric acid may be employed for the purpose of increasing proteolysis (see page 258).

As an anodyne, tincture of valerian or compound spirit of ether is indicated. When, however, the pains are severe, orthoform or anesthesin may be employed; or if so severe as to require the administration of some narcotic, codein or extract of belladonna may be administered in the form of rectal suppositories as well as by mouth. Morphin should be reserved until the final stages of the disease.

Cocain or 3 to 5 minims of chloroform on small pieces of ice may be given for the vomiting.

For distress caused by excessive gaseous fermentation, antiseptic drugs (see page 272) occasionally give good results:

	Gm. or Co.	
R—Resorcinoles,		
Tinctura opii	℥ss 2'0	℥ss
Aquæ	180 0	℥vj
Syrupi	q. s. ad 200 0	℥vij
Misce.		
Sig.—One tablespoonful every two hours.		

Autolysin or *proteal* is the name of a new remedy for carcinoma. Consisting at first of a variety of vegetable proteins, organic salts, extractive materials, chlorophyl, chromophyl, and lipoids, from plant substances little used in medicine, it is now prepared from alfalfa meal, alfalfa seed, and millet seed. The solution contains proteins, proteoses, and peptone, neutralized with sodium hydroxid. The dose is 0.3 to 1.5 Cc. (5 to 25 minims) injected hypodermically into the forearm. The value of the treatment is doubtful.

The medicinal treatment of gastric hemorrhage resulting from carcinoma is the same as that of gastric hemorrhage from other causes (see Chapter XXVI).

Attempts have been made to directly influence the growth of carcinoma of the stomach by means of drugs. Temporary relief has been reported from the use of arsenic in the form of Fowler's solution, administered over a long space of time in gradually increasing doses. Sodium cacodylate may be given in larger doses, 0.1 to 0.5 Gm. ($1\frac{1}{2}$ to $7\frac{1}{2}$ grains) in sterile solution hypodermically. A few clinicians have reported a temporary diminution in the size of the tumors, as well as improvement in the general condition, from the protracted administration of methylene blue. This drug may be given in pill form, 0.06 Gm. (1 grain) three times a day, or in suppositories, 0.06 Gm. with 0.02 Gm. ($\frac{1}{2}$ grain) of extract of belladonna to each suppository. Of other drugs which have been used to inhibit the growth of the neoplasm, or if possible diminish its size, chlorinated soda and chelidonium might be mentioned.

Adamkiewicz has employed a serum called *cancorin*, which is of doubtful value. The carcinoma-cure serum of Doyen is probably also without much merit. Good results in the way of producing a shrinking and softening of the carcinomatous masses have been reported from the use of *cancerodin* (antimeristem), prepared by Schmidt, of Cologne, from killed cultures of *Mucor racemosus*; it causes a febrile reaction and inflammatory manifestations.

RADIATION TREATMENT.—Success in the treatment of carcinoma has been reported from the use of the Roentgen ray; under its influence gastric tumors have wholly disappeared. Einhorn has used radium in the treatment of gastric as well as esophageal carcinomata. The radium is deposited in a hard-rubber capsule, the parts of which are connected by screw threads; the capsule is attached to a silk cord about 75 centimeters in length and introduced into the stomach in the same manner as the stomach bucket (see page 70). There it is retained for an hour at a time. According to Einhorn the results of the radium treatment have been satisfactory, especially in esophageal carcinoma. Radio-active treatment is therefore destined to play an important rôle in the therapeutics of carcinoma of the esophagus, and deserves to be tried on a large scale and in a thorough manner.

In the radium treatment of gastric carcinoma, one great difficulty is to locate the process and place the radium near enough to produce any effect. Powerful as it is, radium cannot destroy the cancer cell at a greater distance than four centimeters ($1\frac{1}{2}$ to $1\frac{1}{2}$ inches). Another difficulty has been lack of standardization of radium itself. Neither of these difficulties is essentially insurmountable.

Treatment of Carcinoma of the Cardia.—The diet should be carefully regulated, to minimize the difficulties of deglutition which accompany stenosis of the cardia, when the seat of the carcinoma is the cardiac entrance to the stomach. It should be such as to produce as little irritation as possible at the cardiac orifice. The use of bland food, soft in consistency, will, to a large extent, ward off the tendency to disintegration of the tumor-like mass, and prevent hemorrhages and rapidity of growth. Meats should be given in finely divided form, and potatoes, vegetables and preserved fruit as purée only. Flour-and-milk soups, eggs, milk, cream and artificially prepared foods may be prescribed so long as they can be swallowed with ease. The maintenance of the general nutrition will not be a difficult matter, since the articles mentioned may be taken in large quantities. As the cardiac stenosis becomes more marked, the question of adequately nourishing the patient assumes a graver aspect. The diet must eventually be entirely liquid.

Patients may partake of the following *ad libitum*: Wine, beer, milk, buttermilk, kefir, fruit juices, mineral waters, vanilla ice-cream, and artificial food preparations. They can be sustained for a long time with the above regimen, providing the painful symptoms are not such as to prevent them from swallowing the liquid nourishment. When patients are unable to consume even liquid diet, owing to difficulties in deglutition, a good quality of olive oil should be administered in generous amount. The oil has the effect of lubricating the stenosed cardia so that food will pass into the stomach more easily. Sometimes the inability to swallow arises more from the inflamed condition of the cardia than from the degree of stenosis; the oil in such cases serves the purpose of a protective layer upon the neoplasm, rendering it less sensitive and thereby preventing spastic contraction of the cardiac orifice. Olive oil, moreover, has a high nutritive value. At least half a wineglass of the oil should be taken morning, noon and night, half an hour before ingestion of other food. Almond milk is the most efficient substitute for olive oil, should the latter become distasteful to the patient. When the stricture becomes so marked as to preclude the passage of even liquids, aided in their passage by the oil, then whatever is partaken of by mouth lodges in the lower portion of the esophagus, above the constriction, only to be expelled by vomiting. Systematic lavage of the esophagus may be performed,

by means of the ordinary soft stomach tube introduced as far as the stricture. When the food remnants are removed the lavage process should be continued with small quantities of warm water until all mucus, pus and blood is washed away. The esophagus may be rinsed with mild antiseptic solutions. After the rinsing process, 30 to 60 Cc. (3j ij) of olive oil should be injected into the esophagus with the esophageal syringe (Fig. 68). Esophageal lavage should be performed at first once a day, and later on every second day. Food may be taken one hour after the lavage. Patients, as a rule, are very much relieved by the systematic washing and lubrication; the irritability of the diseased area is allayed, and frequently deglutition is facilitated. It is possible to improve conditions for a time by having the patients sip every hour a 1- or 2-per-cent. solution of hydrogen dioxid. Some patients are thus restored to comparative comfort and enabled to resume taking nourishment by mouth (see page 272).

By regularly sounding and dilating the cardiac stricture the progress of the stenosis may be inhibited for a considerable length of time (see page 368). Some clinicians, however, are opposed to the use of the sound, fearing mechanical irritation that may stimulate the carcinoma to further growth. It is true that damage may result from the injudicious use of the sound. Mechanical dilatation of the carcinomatous stricture should not be considered so long as the patient is able to swallow a sufficient quantity of liquid and semisolid nourishment to maintain nutrition. When, however, this cannot be done, the physician may succeed in so far dilating the stricture as to enable the patient to swallow with comparative ease. The stomach tube may be utilized for the introduction of nourishment into the stomach. Any food introduced through the tube should be of a concentrated nature, representing the highest percentage of calories per unit of volume. The food may consist of a pint of milk, with somatose or other protein preparation, two or three eggs, three ounces of sugar, malt extract or dextrinized milk, wine and salt. When the feeding by mouth (or tube) is not sufficient, rectal alimentation may be employed for a few days. Analgesic and antispasmodic drugs are sometimes prescribed, to diminish the difficulties of swallowing. Morphine or cocaine may be swallowed as drops or tablets; the following is very useful:

	Gm.	or Co	
R—Morphine hydrochloridi,			
Cocaine hydrochloridi	3R	0'0025	gr. $\frac{1}{2}$
Antipyrine		0 1	gr. iss
Sacchari		0 3	gr. v
Misce et ft. tab. no. 1.			
Sig.—One tablet before partaking of food.			

Solutions of 5-per-cent. cocaine or 3-per-cent. eucain may be injected directly as far as the cardiac orifice by means of a small stomach tube or a long soft-rubber catheter attached to an ordinary piston syringe (Fig. 68, page 354).

SARCOMA.

While carcinoma is a comparatively frequent affection of the stomach, primary gastric sarcoma is rare. Hesch, in 13,387 autopsies, found but six primary gastric sarcomata, and Tilger, in 3500 autopsies, found only one. Although an admittedly rare condition, recent research has shown that many cases diagnosed as carcinoma have upon reinvestigation proved to be of the sarcomatous type. Perry and Shaw, on examining 50 cases of so-called carcinoma ventriculi obtained from Guy's Hospital Museum, London, discovered that four of the specimens were round-celled sarcoma. According to Fenwick, 5 to 8 per cent. of all primary neoplasms of the stomach are to be classed as sarcomata.

Etiology.—Of the etiology of gastric sarcoma very little is known. Heredity and trauma have been considered as positive predisposing influences. Ulcer of the stomach is but rarely the starting-point of sarcoma. Sex seems to have little or no influence as a determining factor. The majority of cases are noted between the ages of forty and fifty years.

Pathology.—Sarcoma is a neoplasm consisting of small cells of an adenoid or embryonic type, without epithelial appearance and in many cases without stroma. We speak of round- or spindle-celled sarcoma, according to the character of the cell. Primary gastric sarcoma occurs in two forms—infiltated and circumscribed. Round-celled sarcomata develop from the trabecular tissue of the gastric submucosa; lymphosarcomata from the lymphatic nodules of the subserous coat. The usual location or starting point of lymphosarcomata is the pylorus; this variety of neoplasm often infiltrates the entire wall of the stomach, but, as a rule, avoids the gastric orifices. Next in frequency is the myosarcoma, having its starting-point in the muscular coat. Fibrosarcomata and myxosarcomata are very rare. Myo- and fibrosarcomata represent the circumscribed form of sarcoma, which often acquires an enormous size, with frequent metastases; especially is this true of round-celled sarcomata and lymphosarcomata, which invade the peritoneal lymphatic glands, the pleural cavities, the kidneys, ovaries, spleen, liver, and lungs. Metastases in the skin are very rare.

Symptoms.—The clinical course of gastric sarcoma is subject to great variation. In some cases symptoms have been present for years, while in others the first dyspeptic symptoms were coincident with a discovery of the tumor. In some cases where the tumor was readily palpable the subjective symptoms were very slight. Cachexia, as a rule, occurs very late in the disease. Owing to the fact that gastric sarcoma seldom produces stenosis, emesis is apt to be absent throughout the course of the disease. Pains in the region of the stomach appear early and may be very severe. Free hydrochloric acid is absent in the majority of cases. Lactic

acid is often found when hydrochloric acid is absent. The Boas-Oppler bacilli are not constantly present in gastric sarcoma. Marked degrees of anemia develop during the progress of the disease. Hemorrhages occasionally take place, though death from hemorrhage is exceedingly rare.

Diagnosis.—The Roentgen ray is of great assistance in the diagnosis. The importance of an early differential diagnosis between carcinoma and sarcoma of the stomach cannot be overestimated, since the timely surgical treatment of sarcoma is frequently followed by gratifying results. Of 26 cases of sarcoma in which resection was done, 11 were reported successful. Lymphosarcomata appear to be especially adapted for operative intervention. The results depend, of course, entirely upon the time of operation.

Many cases of gastric sarcoma cannot be distinguished clinically from gastric carcinoma; especially is this true of the round-celled type. Sarcoma is apt to occur at a much earlier age than carcinoma. Softening, hemorrhage and perforation occur but rarely in gastric sarcoma. Owing to the fact that sarcoma is an infiltrating growth, there is usually no contraction and no obstruction. If obstruction does occur, it is mechanical rather than due to a constriction of the growth.

As an aid to the differential diagnosis the reader is referred to the following:

DIFFERENTIAL DIAGNOSIS.

<i>Gastric Carcinoma.</i>	<i>Gastric Sarcoma.</i>
1 Much pain.	Much pain early, which diminishes as the tumor becomes palpable. Sometimes no pain at all.
2. Involvement of the orifices.	Orifices either not involved or rarely involved.
3. Stenosis marked.	Stenosis seldom.
4. Hemorrhage early.	Hemorrhage late in the course of the disease.
5. Markedly malignant.	Less malignant.
6. Growth rapid.	Growth comparatively slow.
7. Metastases early.	Metastases late.
8 Cachexia early.	Cachexia late.

Treatment.—The treatment of gastric sarcoma is essentially surgical. When for any reason surgical intervention would be injudicious or not likely to be followed by beneficial results, the palliative treatment is that already described for gastric carcinoma.

Coley reports remarkably good results from the hypodermic injection of mixed toxins (the toxins of *Crysipelas* and the *Bacillus prodigiosus*) in the treatment of inoperable sarcoma. The doses are small, not more than $\frac{1}{4}$ to $\frac{1}{2}$ minim to begin with, increased to the point of marked febrile reaction. He states that if benefit is to be expected it will be manifest within three weeks after the toxin treatment is instituted.

BENIGN TUMORS.

Benign tumors of the stomach are of exceedingly rare occurrence. They seldom give rise to any symptoms during life, though occasionally ulceration of the tumor, hemorrhage, or even obstruction may occur. Such growths are, as a rule, discovered at autopsy. They are simple or multiple, sessile or pedunculated. They are classified according to the tissues or gastric layers from which they are derived. Among tumors derived from the glandular structure, or gastric mucosa, are mucous polypi, mucous papilloma and adenoma—small, multiple, sessile or pedunculated growths; individually they are seldom larger than a small bean. They are commonly found near the cardia, rarely in the region of the pylorus.

Tumors derived from connective tissue are: (a) *Lipomata*, or fatty tumors arising from the submucosa in any part of the gastric walls. (b) *Fibromata*. Some of the older writers described these as probably slow-growing carcinomata with much fibroid stroma. To this class belong the fibrous thickenings of the pylorus due to spasm or chronic inflammation or resulting from an old cicatrizing ulcer. True fibromata are villous growths, usually covered with a single layer of cylindric cells; they are often polypoid and pedunculated. (c) *Fibromyomata*—benign tumors which project into the stomach. These consist of unstriped muscle fibers in the fibrous tissue, with the mucous membrane covering intact. They develop in the muscular layer of the stomach wall, are rarely larger than a pea, and produce no symptoms.

Cysts of the stomach are usually formed by the occlusion of a duct of a gastric gland; they may attain the size of a small walnut, but are usually very small and multiple, having the appearance of groups of minute vesicles.

HERNIA EPIGASTRICA.

Hernia epigastrica consists of a rupture occurring at some part of the linea alba between the umbilicus and the ensiform cartilage. It belongs to the class of preperitoneal lipomata, is made up of omentum and fat, and varies in size from a bean to an egg. The regions must be carefully palpated in order to diagnose epigastric hernia; with a tumor of considerable size, there may be felt at the tips of the fingers a sensation as though small shot were hitting them when the patient coughs.

Hernia epigastrica may produce symptoms simulating those of almost any gastric disease, and for that reason it is of the greatest importance that an accurate diagnosis be made. The condition has been mistaken for gastric ulcer, gastritis, gastralgia, carcinoma, enteritis, cholelithiasis, and nervous dyspepsia (see page 419).

One case of mine,¹ reported in 1897, had been previously treated for over four years for a presumed chronic gastritis. There were, with intervals of freedom from symptoms, recurring attacks of nausea, vomiting, epigastric pain, and anorexia. The patient lost 28 pounds in four months on account of his persistent inability to retain food. After the removal of the tumor, which was not known to the patient as hernia, a speedy recovery took place. The patient continued well, without any gastric symptoms, for eighteen years.

The treatment of epigastric hernia is surgical.

¹ Charles D. Aaron, Stomach Disturbances Caused by Hernia of the Linea Alba in the Epigastrium, Medical Record, November 20, 1897.

CHAPTER XXX.

GASTROENTEROPTOSIS.

GASTROPTOSIS; ENTEROPTOSIS; SPLANCHNOPTOSIS; COLOPTOSIS;
NEPHROPTOSIS; HEPATOPTOSIS; SPLENOPTOSIS; CECUM MOBILE;
REDUNDANT SIGMOID.

GASTROPTOSIS (a term for which we are indebted to Glénard) is so frequently complicated with displacement of the intestine and other abdominal organs that it may be considered conveniently under the heading of Gastroenteroptosis. Separate names have been given to the downward displacement of abdominal viscera, among which we have gastrop^tosis, referring to downward displacement of the stomach; colo^pto^sis, or downward displacement of the colon; hepato^pto^sis, spleno^pto^sis, nephro^pto^sis, entero^pto^sis, referring respectively to downward displacement of the liver, spleen, kidney, and intestine. The etiology and clinical manifestations of these conditions, as well as their treatment, are so similar that they may be considered with advantage together.

Etiology.—Gastroenteroptosis is a condition frequently met with in patients who consult a physician in regard to digestive disturbances. It is a disease of comparatively young adult life, appearing soon after puberty, but is occasionally met with in patients over fifty years of age. Females are particularly disposed to gastroenteroptosis.

Forms.—From the viewpoint of etiology two different forms of gastroenteroptosis are to be distinguished. The first is the result of causes acting mechanically, the principal etiologic factors being improper modes of dress, traumatism, frequent childbirth, and tight lacing; all these causes are aided by poor nutrition and severe physical toil. This form of gastroenteroptosis comprises a comparatively small number of cases. Frequent pregnancies, by bringing about a condition of relaxation of the abdominal wall and diastasis of the recti muscles, producing thereby a pendulous abdomen, are responsible for many of these cases of gastroenteroptosis. It has been shown repeatedly that tight lacing is productive of downward dislocation of the intestine. The removal of large abdominal tumors and frequent paracenteses in order to free the abdomen of ascitic fluid, are also etiologic factors. Organic diseases in general may likewise lead to ptosis of the stomach. The causal relation between trauma of the abdomen and displacement of the kidney is well known.

The second form of gastroenteroptosis is due to a constitutional hereditary predisposition. Thanks to the researches of Stiller, more is understood of this variety of downward displacement than formerly, and his views are now almost universally accepted. According to this writer, in 90 per cent. of cases of gastroenteroptosis the abnormal position of the abdominal viscera is quite a



FIG. 87.—Habitus enteroptoticus (asthenia universalis congenita).

distinct form of the physical conformation. Stiller calls it "habitus enteroptoticus" or "asthenia universalis congenita" (Fig. 87). Patients suffering from this weakness and from the characteristic bodily form are apt to develop into neurasthenics. The condition of gastroenteroptosis is often complicated with gastric and intestinal atony and nervous dyspepsia. A well-marked case of habitus enteroptoticus presents a complex of symptoms, namely, those of gastroenteroptosis, gastric atony, and nervous dyspepsia. This complex of symptoms has been designated general asthenia. These three affections are not always present, however, in the same degree of intensity; gastroenteroptosis gives rise, as a rule, to the most pronounced symptoms. The mechanical causes mentioned above would not, in all probability, give rise to gastroenteroptosis were the patient not predisposed to this condition by the habitus enteroptoticus. Hence it may be concluded that gastroenteroptosis from purely mechanical causes is a rare condition.

Pathology.—Gastroptosis does not imply a dropping or downward displacement of the entire stomach. On account of the attachment of the stomach at the cardia it is impossible for the displacement to be complete; when we speak of gastroptosis a descent

of the pylorus and that part of the stomach directly in front of the vertebral column is implied. With the downward displacement of the pylorus there is likely to be a stretching of the stomach from the cardiac orifice toward the pylorus. This forms the so-called "water-trap" stomach (Plate XIII, Fig. 1). Several abnormal conditions enter into the development of both gastroptosis and

enteroptosis. The ligaments and mesenteries may become relaxed, thus permitting a displacement of the organs attached to them; or the intra-abdominal equilibrium may be disturbed by alteration of the intra-abdominal pressure upon which it depends, and gastro-enteroptosis results.

Part of the duodenum as well as the entire large intestine may be displaced. The mesenteries easily stretch to meet this condition. Certain mesenteric supports of the intestine are more rigid than others and will not yield to the displacement. The ligaments at the hepatic and splenic flexures of the colon do not yield readily, and under traction these parts become abnormally kinked, simulating stenosis. In this condition the intestinal contents pass through with great difficulty. The distal end of the duodenum is fixed by a strong ligamentous band to the diaphragm. The duodenum, being fixed, cannot move down, while all the other abdominal organs glide down during gastroenteroptosis. Hence the transverse segment of the duodenum becomes compressed by the superior mesenteric artery, inducing duodenal dilatation (Plate XVII, Fig. 2, opposite page 144). The downward displacement of the stomach tends to produce stenosis of the fixed part of the duodenum, with chronic dilatation. The symptoms of chronic dilatation of the duodenum are: persistent or recurring vomiting of bile, pain referred to the right hypochondrium, constipation, headache, and nervous symptoms. In marked cases starvation with acidosis develops and leads to a fatal termination.

The transverse colon, owing to its ligamentary connection with the stomach, is most frequently involved. Any material degree of gastropptosis causes coloptosis (Plate XVIII, Fig. 1, opposite page 144), since the transverse colon is made to descend. The hepatic flexure suffers in gastroenteroptosis, because as the stomach passes downward it forces the transverse colon before it and hence makes more and more acute the hepatic flexure (Plate XVIII, Fig. 3). However, since the hepatic flexure is usually obtuse or rectangular in outline, it is seldom drawn so tightly by the hepatic ligament as to cause very pronounced stenosis. The splenic flexure forms normally an acute angle; it is a distinctive anatomic provision for fixing the colon to the costal wall. In gastroenteroptosis the splenic flexure is dragged on and its angle made more acute (Plate XVIII, Fig. 2). The stomach drags the middle of the transverse colon downward, which tightens the hepatic and splenic flexures in direct proportion to the extent of the gastropptosis (Fig. 88).

A prolapsed cecum may pull on the end of the ileum, while the short, firm mesentery of the latter acts as a counter-pull, inducing the Lane kink of the ileum (Fig. 89, *b*). The dropping of the lower coils of the ileum will cause the same kink. This kink does not occur if the abdominal viscera are in their normal position. The form of attachment of the cecum makes it freely movable.

An abnormal mobility of the cecum of two to four inches has been emphasized by Wilms (see *Cecum Mobile*, p. 146). Occasionally the cecum fails to rotate; in this congenital malformation the terminal coils of the ileum are posterior to the cecum (see page 770).

The sigmoid flexure, the most movable part of the large intestine, is likewise liable to undergo important positional changes. Just as its length and lumen may experience widely varying deviations

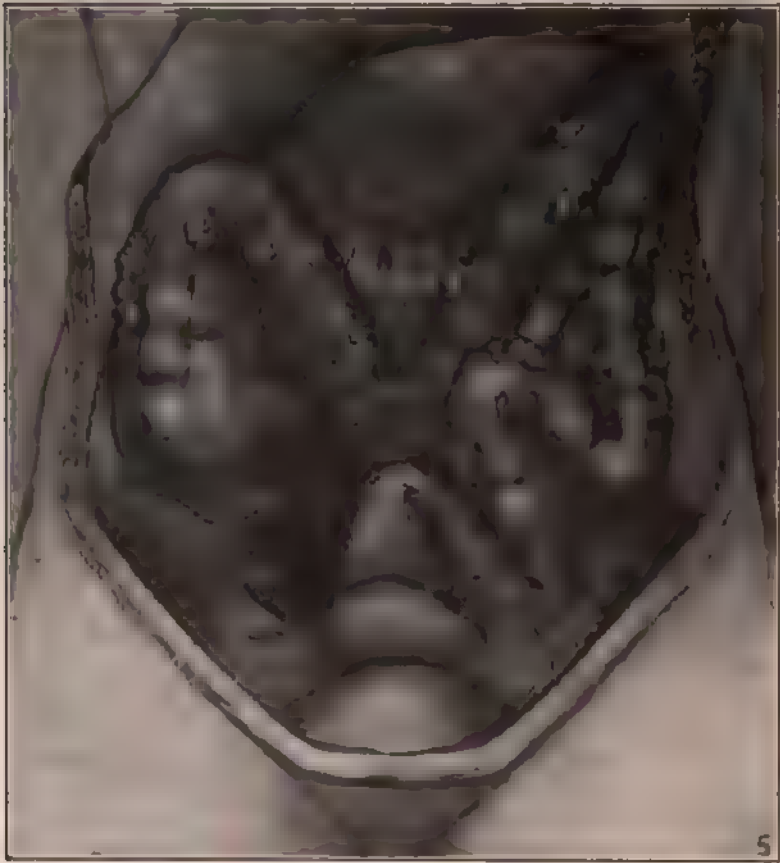


FIG. 88.—Descent of transverse colon, acute angulation at the splenic flexure, with adhesion and angulation of the sigmoid flexure. (Tuttle.)

from the normal (Hirschsprung's disease), so may its position and loop-formation present equally varying abnormalities (redundancy).

Glénard, the apostle of gastroenteroptosis, describes a condition which he calls *phrenoptosis*—that is, downward displacement of the diaphragm. He considers it responsible for the condition known as *movable heart* or *cardioptosis*, though itself merely an episode of the general tendency to ptosis.

At certain points kinks are apt to occur when there is a dropping of the stomach and intestine (Fig. 89). These may all be easily made out by the use of bismuth and the Roentgen ray (see Chapter V). From above downward, kinking may occur at the junction of the duodenum and jejunum (Fig. 89, *a*), at the distal end of the ileum (Fig. 89, *b*), at the hepatic flexure (Fig. 89, *c*), at the splenic flexure (Fig. 89, *d*), and at the sigmoid flexure (Fig. 89, *e*). Should pericolic membranes form around these kinks, an actual obstruction may occur.

During embryonic evolution certain membranes form and disappear. In certain instances, for some untold reason, these temporary membranes remain as a thin veil (Jackson's), containing

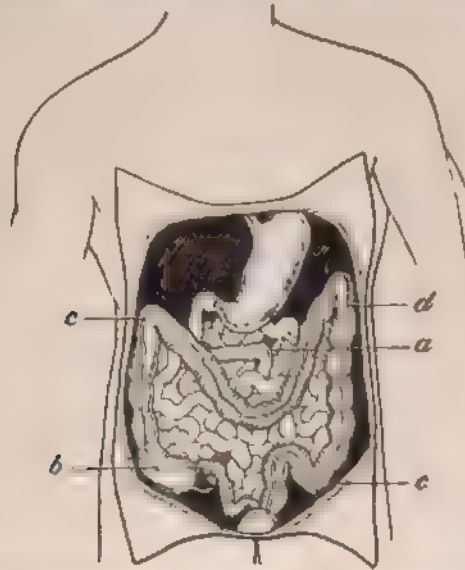


FIG. 89. Kinks of the intestine. *a*, duodenum, *b*, ileum, *c*, hepatic flexure, *d*, splenic flexure, *e*, sigmoid flexure

small linear vessels. These pericolic membranes are not pathologic; they do not do any harm. Should an alimentary toxemia develop, however, *pericolicitis* results and the membranes become thickened and obstructive. This pathologic change often occurs in gastroenteroptosis, producing angulations, kinks, adhesions, bands, narrowing, and chronic intestinal stasis (see Chapter XXXIX).

Recent experimental work by Keith and his discovery of nodal tissue in the intestine (see page 64), explaining the mechanism of intestinal movements, sheds a new light upon the causation of intestinal stasis. Notable quantities of this "nodal and conducting" tissue were found in the region of the cardia of the stomach,

where it initiates gastric movements; near the ampulla of Vater, to control the movements of the pylorus and duodenum; a lesser quantity near the beginning of the jejunum, exercising the same function over the greater portion of the remaining small intestine; in the region of the ileocolic valve, to control the lower ileum and proximal portion of the colon; and also in the transverse, descending and iliac colon, and in the rectum. Not only do the anatomic sites and the demonstrable physiologic functions of these "nodes" explain the normal movements of the intestine, but it is obvious that a perversion of the function of any one of them is capable of giving rise to an inhibition of the forward progress of the intestinal contents, with resulting intestinal stasis. In the establishment of this as the physiologic explanation of the mechanism of the production of intestinal stasis, Keith was able to demonstrate the presence of definite fibrotic and degenerative changes in this nodal tissue in segments of the intestine removed for the relief of chronic intestinal stasis (see page 696).

Symptoms. Many patients with habitus enteroptoticus which has developed into pronounced gastroenteroptosis do not experience any troublesome symptoms whatever. The same may be said of those whose gastroenteroptosis is the result of purely mechanical causes. On the other hand, many patients have been relieved of the distressing symptoms accompanying gastroenteroptosis without correction of the anatomic displacements. It may be inferred from this that gastroenteroptosis in itself does not produce any marked disturbance or discomfort to the patient. The constitutional neurasthenia of enteroptotics is responsible for a great many of the subjective symptoms ascribed to gastroenteroptosis itself. The displacement merely aggravates the neurasthenic effects, or perhaps in some instances initiates them by the continuous traction of the displaced viscera on the mesenteries, thus placing the abdominal sympathetic nervous system in a condition of continued reflex irritation. Atony of the stomach and intestine, a frequent accompaniment of well-marked ptosis of these organs, is productive of many untoward symptoms. Patients complain of a variety of nervous manifestations, such as lassitude, dull headache, inability to work, mental depression, and general weakness. The gastric symptoms consist of pressure, fulness, nausea, and belching; occasionally pain is felt in the region of the stomach. Indications of nervous dyspepsia (see Chapter XIX) are also in evidence as burning sensations in the stomach, hyperacidity, and vague discomforts after eating. The appetite is, as a rule, poor, though on rare occasions patients have ravenous appetites. Gastroenteroptosis is often accompanied, in women particularly, by severe backaches. These enteroptotic conditions are closely related to persistent constipation. It is easily intelligible, and has also been clearly pointed out, that in consequence of these conditions,

especially in the presence of constipation, catarrh may occur, showing a predilection for the flexures and the sigmoid. In a clinical respect it is therefore sufficient to refer the reader to the chapter on Constipation (Chapter XXXVII).

Objective Symptoms.—The objective symptoms in cases of well-marked habitus enteroptoticus are very characteristic. The patients, as a rule, are tall in stature, with long arms, thin neck, narrow elongated thorax, long flat abdomen, and a predisposition to flat-foot and lordosis. Children are able to subluxate the first phalanx of each finger on the metacarpal of the preceding one. The habitus enteroptoticus impresses the observer as being similar to the habitus phthisicus. The bony structure is slight, the muscles weak, and there is a marked diminution in the adipose tissue which gives grace to the physical appearance. Enteroptotic patients, as a rule, look pale and give the impression of being ill. Their spirits are usually depressed. Characteristic alterations of the thorax belong also to the habitus enteroptoticus: the thorax is long and narrow and the shoulders slant downward. The epigastric angle is markedly acute. The intercostal spaces are sunken and the abdominal walls are thin and flaccid. The distance between the umbilicus and the ensiform cartilage is greater than in a normal person. The Lénhoff index—the distance in centimeters from the episternal notch to the symphysis pubis, multiplied by 100, and divided by the greatest circumference of the abdomen—is over 80 centimeters. The epigastric region is sunken when the patient stands erect. The abdomen, however, below the navel protrudes in consequence of the weight of the descended abdominal viscera (Fig. 87). There are frequently found broad spaces between the recti muscles (diastasis). During respiration the lesser curvature of the stomach will be discerned at times and may be outlined beneath the thin abdominal wall. A special feature of the habitus enteroptoticus is a movable tenth rib, which is shortened and easily displaced in consequence of the absence of the cartilaginous attachment. This fluctuating rib, known as Stiller's sign, is present in about 70 to 80 per cent. of all cases of gastroenteroptosis. In female patients it is possible at times to palpate the abdominal aorta and to ascertain strong pulsation on but slight pressure. The ease with which the abdominal aorta may be palpated is a sign of neurasthenia, and this condition is due to a dilatation or paralysis of the vessel wall brought about by reflex causes. The celiac plexus, which is located on the anterior surface of the abdominal aorta in the epigastric region, is not infrequently very sensitive to pressure; this condition is also considered suggestive of neurasthenia.

A high pulse-rate when the patient is standing is the rule in cases of gastroenteroptosis. The difference between the pulse-rates in the standing and recumbent positions is an index of the intensity

of the abdomino-cardiac reflex. The abnormal acceleration of the pulse-rate is supposed to be due to the dragging of the stomach and intestine on the fibers of the vagus.

Diagnosis. *Gastroenteroptosis* is recognizable by means of inflation, auscultatory percussion (Benedict), and transillumination. The value of Roentgen-ray examination lies in its detection of the true state of affairs (see Chapter V). The diagnosis of all these dislocations can be absolutely confirmed by this method of examination.



Fig. 90. Locating the point of tenderness.

In cases of gastroenteroptosis, deep continuous pressure with the ends of the fingers over the celiac plexus in the epigastrium will induce pain (Fig. 90). The point of sensibility varies in different individuals. To locate it, the cooperation of an assistant is necessary. With the patient standing, the physician applies his fingers in a series of deep pressures until the point of greatest tenderness is found. The fingers are held at this point. The nurse then takes a position behind the patient, passes both arms about him so that the hands, meeting in front, rest on the hypogastrium, and lifts the abdomen in its entirety (Fig. 91). This relieves the epigastric pain at once, despite the great pressure

exerted by the physician at the point of tenderness. When, however, the nurse allows the patient's abdomen to drop to its former position, the deep pressure continuing, the pain reappears (Fig. 90).

This sign is constant in gastroenteroptosis.¹ In organic disturbances, such as gastric ulcer, carcinoma, etc., the pain under pressure continues even when the abdomen is lifted.

The pain on pressure, relieved by lifting the abdomen, is an objective sign which I believe to be reliable in the diagnosis of gastroenteroptosis.



FIG. 91.—Nurse lifting abdomen.

Nephroptosis. Displacement of the kidneys is frequently found in gastroenteroptosis is often, indeed, a pathognomonic sign. The right kidney is usually the one affected. The terms movable kidney, dislocated kidney, wandering kidney, floating kidney, prolapsed kidney and nephroptosis have been applied to a variety of renal displacements. Movable kidney is said to be five or six times more frequent in women than in men. Both kidneys movable is a condition observed almost exclusively in women. Inasmuch

¹ Charles D. Aaron, A Diagnostic Sign of Gastroenteroptosis, *The Archives of Diagnosis*, New York, April, 1917.

as movable kidney implies gastroenteroptosis, it is of the utmost importance to diagnose the condition. The diagnosis is easily made by palpation. The correctness of the result depends, of course, on the degree of technical skill applied in manipulation. Every physician can acquire the art of palpation by careful study and practice. One hand is placed on the back, over the lumbar region, and the other on the abdomen; bimanual palpation is always necessary. The clothing should be removed and the palpating hands brought in direct contact with the skin. The abdomen of the patient should be relaxed as completely as possible before the examination. The hands of the physician should be placed flat, one on the back and one on the abdominal wall. Severe pressure with the fingers should be avoided. It is best to begin softly, allowing the pressure to become gradually greater. The palpating hands should be warm, since cold hands cause contraction of the abdominal muscles and prevent deep manipulation. In cases where the tension of the abdominal walls is too great, chloroform narcosis may be employed. This is, however, rarely necessary. The physician may often feel the kidney slide from under his hands; its smooth surface and distinct outline are very characteristic. In palpating for movable kidney the patient is placed in three different positions:

1. Standing while the manipulator sits on a chair.
2. Lying on the back while the manipulator sits on the edge of the couch.
3. Lying on either side, according to which kidney is being palpated, while the manipulator sits.

First position (Fig. 92). This is the most important position for palpating a movable kidney, since it permits the maximum displacement, and the kidney is therefore easily felt. Begin by superficial pressure, and later use deeper manipulation. Superficial pressure reveals the resistance in the abdomen while the abdominal muscles support the viscera, and the hands soon differentiate between the natural and the artificial support of these muscles. Deep palpation in this position is of great importance, since frequently the kidney can be held in the hand. With one hand on the lumbar region the whole abdomen must be explored with the other, as a movable kidney may be displaced anywhere from its normal position, even as low as the symphysis pubis. The peculiar shape of the kidney, its smooth characteristic feel, and the way it slips from the hand under the ribs will make it easily recognizable.

When a kidney is in normal position it moves slightly during respiration. A normally located kidney cannot be palpated. When one-third of the kidney can be palpated the condition is spoken of as displacement of the first degree; when one-half is palpable, displacement of the second degree; when the whole

kidney is palpable, displacement of the third degree. The same procedure should be followed out in palpating the kidney on either side. On account of their close attachment to the diaphragm, the liver and the gall bladder move during respiration. Care should be exercised lest they be mistaken for the kidney.

Second position (Fig. 93). In this position the patient lies on his back, with the shoulders raised and the legs slightly flexed. One hand of the physician is placed on the lumbar region and the other flat on the abdomen, below the costal margin, along the



FIG. 92. First position for palpating movable kidney.

outer border of the rectus muscle. The patient should be instructed to take a deep, slow inspiration, when the kidney, if movable, may be felt between the hands. The kidney naturally drops back to its normal position when the patient lies on his back, for which reason it is wise to resort to other positions in order to confirm the diagnosis. Usually mobility of the third degree is best made out with the patient in this position.

Third position (Fig. 94). The patient should lie upon the side opposite to that to be explored. The shoulders should be thrown

forward and the thighs slightly flexed. The physician should sit on the edge of the couch. One hand over the lumbar region and the other over the abdomen will bring the kidney between the two hands. To bring it lower, should it be movable, the patient is instructed to take a deep inspiration, when the diaphragm will

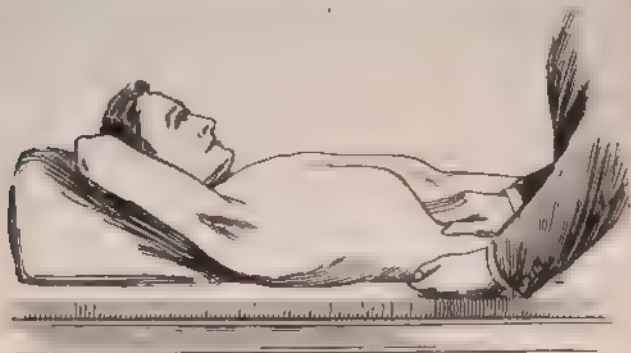


FIG. 93.—Second position for palpating movable kidney.

force it downward; then during expiration it can be held firmly between the hands. The slightest relaxation of the pressure of the hand will permit the kidney to slip away from between the fingers, which is characteristic of no other organ.

Hepatoptosis. Hepatoptosis, dislocation of the liver, is of frequent occurrence, and when overlooked may give rise to diagnostic

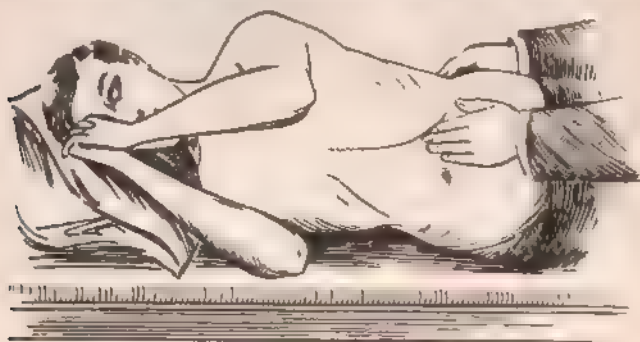


FIG. 94.—Third position for palpating movable kidney.

error. Hepatoptosis originates from the same general causes as gastroenteroptosis. Glénard found in two-thirds of his cases of hepatoptosis that nephroptosis also was present. Hepatoptosis with hepatic colic is frequently mistaken for cholelithiasis (see page 605).

Splenoptosis.—Abnormal positions of the spleen are rarely found, except occasionally when it is enlarged.

Prognosis.—The prognosis for permanent replacement of the displaced organs is, as a rule, not good. A ptotic stomach or intestine remains so. The distressing symptoms accompanying the condition may, however, be entirely removed or greatly ameliorated, so that patients with congenital habitus enteroptoticus may pass the remainder of their lives in comparative comfort. We cannot give these patients normal mesenteries, but we can give them normal function.

Prophylaxis.—Prophylaxis, so far as the mechanical causes of gastroenteroptosis are concerned, consists in keeping patients in bed for a sufficient time after parturition, reinforcing the abdominal muscles by abdominal bandages, and strengthening the muscles by exercise and massage. By these measures much of the muscular relaxation of the abdominal wall following childbirth may be avoided. Properly fitting corsets are a valuable prophylactic agency. The habitus enteroptoticus may sometimes be recognized in young subjects by their peculiar physique and weak stomach. In such subjects marked departure from the normal may be retarded by suitable preventive treatment in spite of the existing predisposition.

Treatment. The treatment of gastroenteroptosis should be directed toward improvement of the general nutrition, in order to counteract the neurasthenia and to strengthen the muscles of the abdominal walls.

Patients who are poorly nourished must be well fed. The diet should be as nutritious as possible; it should contain a large proportion of fat. Milk, cream and butter are among the most suitable articles of food for this condition. The nutrition must be governed almost entirely by the requirements of the individual case. The motor and secretory powers of the stomach should always be considered in prescribing diet.

Hyperalimentation.—Sometimes it is necessary to resort to "forced feeding," by which we mean hyperalimentation. It is well, however, before attempting systematic hyperalimentation, to ascertain the actual powers of assimilation of the patient. In determining the status of a patient's nutrition two factors must be borne in mind—first, the condition of the protoplasm (muscles and blood); and secondly, the amount of fat present. The protoplasm is estimated from the muscular mass. A person with weak muscles, as a rule, suffers from deficiency in nutrition. An attempt should be made to strengthen the weak muscles of these patients by hyperalimentation, and thus bring about an improvement in the quality of the blood.

Fat should constitute 18 to 20 per cent. of the total body weight of the adult male, and 25 to 28 per cent. of the weight of the female.

It is necessary, then, for the physician to estimate as well as he can the quantity relation between adipose tissue and muscle. In certain diseases the presence of what might be termed an excess of fat is not an undesirable feature, while in other ailments it is desirable that the amount of fat be less than in the normal individual. In gastroenteroptosis and neurasthenia it has been found advisable to keep the nutrition up to the highest possible point, and that patients do better when the amount of adipose tissue is above the indicated percentage for their body weight. In pursuing a course of hyperalimentation it is an advantage to know the quantity of nutriment required by each patient in order to maintain his particular body weight. The following values have been calculated for this purpose.

The patient requires:

	Calories per kilogram body weight for the twenty-four hours
1. When kept in bed	30 to 35
2. When confined to the room	32 to 35
3. When employed at light labor	35 to 40
4. When employed at moderate physical labor	40 to 45
5. When employed at hard labor	45 to 50

A diet corresponding to the above table is designated a "sustaining diet." Such a regimen, it will be seen, will vary in the same individual, depending upon the question of rest or physical activity.

Before beginning the so-called hyperalimentation cure it is necessary to ascertain the sustaining diet for the patient. This may be easily accomplished by referring to the standard tables of food substances, which give the exact percentages of protein, fat and carbohydrates in the food, with the caloric value of each (see Chapters VI and VII). The calculation of the food value of dishes complex in composition should be entered upon with great care. An exact knowledge of the composition and food value of soups and farinaceous foods is necessary if the physician is to avoid error in dietary prescription. When it is desired to ascertain the exact condition of undernutrition of a patient, the food should be carefully weighed and estimated in calories and the result compared with the sustaining diet of that particular patient. Should the amount of food ordinarily ingested by the patient be less than the sustaining diet, the condition is one of undernutrition.

Hyperalimentation consists in the ingestion of certain quantities of nutritive material in excess of the amount of the sustaining diet—the intention being, of course, to increase the weight of the patient. This added nutriment is known as the food surplus.

Von Noorden has calculated the probable increase in weight during a course of hyperalimentation as follows.

Daily increase in food	Weekly increase in weight
500 to 800 calories yield	600 to 1000 Gm.
800 to 1200 calories yield	800 to 1200 Gm.
1200 to 1800 calories yield	1200 to 2000 Gm.

Of the total number of calories represented in the added food in a course of hyperalimentation, 8 per cent. is used up for purposes of digestion and assimilation; about 4 per cent. is lost in the feces; and 10 per cent. is stored up as protein. The remaining 78 per cent. is assimilated as fat.

One of the best means for increasing the amount of muscle tissue is systematic muscular exercise. If the patient can be persuaded to take regular muscular exercise during the food cure, a marked increase in flesh will result. It has been demonstrated that muscular activity develops the muscles. This would seem to be in opposition to the food cure as outlined by those who first made use of it. Weir Mitchell and Playfair insisted upon having their patients maintain the recumbent position. But one's cases must be differentiated. Some are too weak for exercise, or the condition of the digestive system may require absolute quietude.

It is, moreover, advisable that every patient be put to bed for the first eight days at least when undergoing the so-called food cure. This will accustom him to the regular administration of food and likewise reduce the combustion processes to the lowest possible degree. The radiation of heat is diminished and its retention favored by complete rest. Many writers, however, prefer that patients should undergo active muscular movement as soon as there are signs of increase in weight during the first week. The slight loss of weight which may result from this muscular exercise is soon compensated by the marked increase in appetite which follows the bodily activity. The muscular exercise should be so arranged as to avoid undue fatigue. The condition of a patient after a course of hyperalimentation combined with muscular exercise will be much more vigorous than if the exercise had been omitted.

Technic of Nutrition. The diet should not consist of protein substances alone. Their caloric value is more than offset by the difficulty with which they are assimilated. From 12 to 15 per cent. of the energy afforded by a protein diet is lost in digestion, as compared with the 8 per cent. waste from a mixed diet. Protein increases combustion. In the sustaining diet the daily quantity of protein is about 100 Gm.; in hyperalimentation it should be between 100 and 120 Gm. This amount of protein is found in the ordinary mixed diet. Protein may be administered in any form, such as the lean varieties of meat, fish, or fowl. Fat has the disadvantage, as compared with lean meat, of more quickly satisfying the appetite or exciting a distaste for animal food. The portion of meat should not be so great as to prevent the ingestion of other nutriment. Nervous patients should not receive too much meat; for this class of patients, eggs should be freely prescribed, as well as cheese and milk. The artificial protein preparations are also worthy of consideration here. Casein preparations are espe-

cially useful in cases where the amount of food the patient is able to take is small. (See Chapter VIII.)

Fat, as already intimated, is the most valuable article of diet in the food cure, owing to its very high index of combustion. At least 180 Gm. (6 ounces) per day should be tentatively prescribed. Patients often consume with ease as much as 250 Gm. (8 ounces) of fat during the twenty-four hours. It is well to have some standard as to the quantity of fat to be consumed daily during the course of the food cure or hyperalimentation. Von Noorden suggests the following daily regimen:

200 Gm. butter	=	160 Gm. fat	=	1400 fat calories
1 liter of milk	=	33 Gm. fat	=	307 fat calories
300 Gm. cream	=	75 Gm. fat	=	698 fat calories

The total amount of calories yielded by the fat in this diet is 2495. Not every patient is able to partake of this quantity of fat. Cream in such large amount is apt to cause disturbances which destroy the appetite. It is possible, nevertheless, in constructing a dietary with fat as a fixed basis, to attain a high caloric value. Fat should be prescribed either as liquid or in a form easily reduced to liquid, such as butter, milk, yolk of egg, rich cheese, and chocolate. The resourceful chef or housekeeper will find many ways in which these articles may be worked up into a variety of tasty dishes. Butter may be taken by itself or may be made an ingredient of gravies, so that as much as 200 grams (7 ounces) per day may be easily ingested. The caloric value of milk may be increased by adding cream. Coffee, tea, or milk soups may be administered with milk, according to the taste. Kefir and yoghurt are also useful (see page 164).

The carbohydrates, owing to the fact that they permit of rich variety in food, form important elements in the food cure. They also render unnecessary the prescribing of much protein. Carbohydrates in the hyperalimentation cure are capable of being absorbed to the amount of 180 Gm. (6 ounces) per day. They may be used as vehicles for butter, eggs, or milk. The carbohydrate carriers usually employed are wheat bread, biscuits, zwieback, milk soups, oatmeal, cereals, and breakfast foods. Thick soups are best taken early in the morning and during the evening meal rather than at noon, owing to their satiating qualities. Oatmeal porridge or hominy may be eaten with cream. Vegetables such as potatoes should be given in the form of purée with large quantities of fat. Many patients are particularly fond of chocolate, which may be taken with or without cream. Sugar and fruit juices may be prescribed. Unfermented grape juice, which is very agreeable to the patient, may be prescribed. The malt preparations, such as malt extract, are acceptable to many patients; they possess some carbohydrate value.

Alcohol is considered by some authorities as possessing food

value. It is, however, of but little importance as an element in the food cure. Undoubtedly the effect of alcohol upon the nervous system more than offsets any virtue it may possess as a food.

All dietary regulations should be made with due regard to the secretory and motor conditions of the stomach according to the directions laid down in the chapters dealing with secretory and motor derangements. Before a food cure is instituted it is necessary to know whether achylia, subacidity, normal acidity or hyperacidity is present, and also the condition of gastric motility.

Under favorable conditions the food cure may be carried on at the home of the patient. Better results, however, are obtained when patients are prevailed upon to leave their home surroundings and enter a well-managed hospital or a sanitarium where special attention is given to the dietetic treatment of disease. Much is achieved with this class of patients by surrounding them with salutary mental influences. The mental influence which the physician may be able to exert over his patient has an important bearing upon the success of the treatment. An endeavor should be made to inspire the patient with hope, and to overcome as far as possible his prejudices. Once the patient's weight begins to increase, and hope and confidence are established, there are usually few if any serious difficulties to overcome. The patient should be educated to appreciate the nature of his disease; he should understand that improvement will be gradual and will depend largely upon his habits of living and his mental attitude for its permanency.

As the general nutrition of the patient improves, the stomach and intestine will likewise become tolerant of a greater quantity and variety of food. Patients with gastroenteroptosis complicated with neurasthenia should be considered cured only when they may again partake of a normal diet without any distressing after-symptoms and when the work of the intestinal tract is normally performed. To accomplish this result is the purpose of the food cure.

Hydrotherapeutics.—Hydrotherapeutic measures may be instituted and carried out in conjunction with the food cure (see Chapter XII); they should be limited, however, to methods of a stimulating and invigorating character. In asthenic conditions of the heart muscle, systematically performed respiratory gymnastics are to be carried out several times a day. The muscles of the body may be stimulated by dry rubbing of the skin with rough towels. The hydrotherapeutic procedures suitable to gastroenteroptosis complicated with neurasthenia consist in the application of cold water, half-baths, Scotch douches on the abdomen and stomach, cold friction, rubbing and slapping, and cold full packs. The prolongation and intensity of these hydrotherapeutic measures must be varied to suit the requirements of the case. Nervous debility will at times be greatly benefited by sojourn in the country, at the seashore, or at some other climatic health resort.

Massage and Exercise.—Massage plays an important rôle in strengthening the abdominal muscles. For details as to the method of procedure, see Chapter X on Massage. In addition to the massage the patient should perform gymnastic exercises to invigorate the abdominal wall. He may assume a squatting posture, with the knees flexed until the thigh rests on the calf; or he may be instructed to raise himself into the sitting posture when lying flat upon the back. The patient should be taught to lie at full length in order to counteract atony of the gastro-intestinal tract by a better circulation of the blood. If a bag of salt weighing from one to five pounds be placed on the abdomen, the efforts of the patient to elevate the abdomen with this weight upon it will strengthen the muscles.

The massage process in gastroenteroptosis must be varied according to the anatomic relations of the parts. Gastric massage may immediately precede abdominal. When these movements cannot be conveniently performed daily by the physician, the patients may practice on themselves by means of a cannon ball, which should not weigh more than from three to five pounds. A sphere of wood weighted with shot answers the purpose in automassage very nicely.

Electrotherapeutics. Gastroenteroptosis is sometimes improved under a course of electrotherapeutics. In relaxation of the abdominal muscles and intestinal torpor, faradization is indicated. Two large plate electrodes four to six inches square are applied to the two sides of the abdomen, or, if desired, over the epigastric and hypogastric regions. The faradic current should be turned on slowly and its strength increased gradually so that distinct contraction of the abdominal muscles becomes apparent. The galvanic current is indicated in cases characterized by abdominal pains of neurotic origin. As much as thirty milliampères may be used, with one electrode over the stomach and the other over the bladder (see page 214).

Mechanical Treatment of Gastroenteroptosis.—The mechanical therapeutics consists principally in the bandaging of the abdomen with a view to supplying support to the relaxed abdominal wall and to fixing the displaced viscera. The treatment is merely palliative, but of very great value. It acts beneficially by ameliorating the symptoms which arise from tension or stretching of the mesenteries.

The mechanical support consists of abdominal bandages or abdominal corsets. Apparatus for this purpose is available in great variety, but everything has its peculiar defects, such as uncomfortable perineal straps or badly fitting pads, which occasion patients no small degree of annoyance. It is a very difficult matter to find well-fitting "ready-made" abdominal bandages or corsets. Among the best known and most suitable appliances

for the treatment of gastroenteroptosis are the abdominal bandage of Glénard and the ordinary silk-rubber abdominal bandage used for a pendulous abdomen. The latter possesses great adaptability and is light in weight.

In order to ascertain whether a bandage is indicated in a given case, the so-called "belt sign" of Glénard should be employed. The physician, standing behind the patient, passes his arms on either side and places both hands on the lower abdominal wall. With the hands in this position the abdominal mass just above the pubes can be easily raised. The physician should then suddenly remove his hands, permitting the abdominal mass to fall; if then the patient's distressing symptoms, relieved by the temporary support, return, the indication is positive for the use of an abdominal bandage or support. Glénard calls this phenomenon "*épreuve de la sangle*" (Fig. 95). Should the patient experience no relief



FIG. 95. Glénard's "belt sign" (*épreuve de la sangle*).

when the abdomen is lifted, and feel if anything better when it is permitted to assume its old position, the bandage will not give good results.

The Aaron bandage,¹ as presented before the American Medical Association at Philadelphia, June, 1897, has been used by me with success in selected cases (Figs. 96 and 97). It has in no way been modified from the form introduced to the profession, and has proved eminently satisfactory. It is supplied with a truss which is fitted to the bandage encircling the body below the crests of the ilium and above the trochanter (Plate XXIV). This band-

¹ The bandage devised by the author is manufactured by G. J. De Garino Co., 33 West 42d Street, New York.

age exerts a pressure upon the hypogastrium from below upward, raising the intestine, which in turn acts as a cushion for the stomach. In this way the tension upon the abdominal organs is relieved. The bandage should be adjusted properly in order to prevent slipping up at the back, or no benefit will be derived from its use (Plate XXV, Fig. 2). When properly applied it affords abdominal support and at the same time leaves the ribs and diaphragm free from all compression and the respiratory movements free. There is no pressure over the solar plexus. The wearing of this bandage tends to develop in patients a deep, broad, prominent lower chest and epigastrium, which is the condition found in well-developed normal individuals (Plate XXV, Fig. 1).



FIG. 96. Author's abdominal bandage.

This bandage has been found valuable in the treatment of abdominal pain due to a loose sacro-iliac joint. This pain sometimes extends through to the front of the joint, where it is most apt to be mistaken for an affection of some pelvic organ. The existence of this condition is, as a rule, indicated by the fact that the affected leg is shortened, and that pain results when the leg is rotated, forced strongly, or pulled outward. Most of these patients are relieved by the application of the bandage.

PLATE XXIV



Abdominal Bandage in Position.

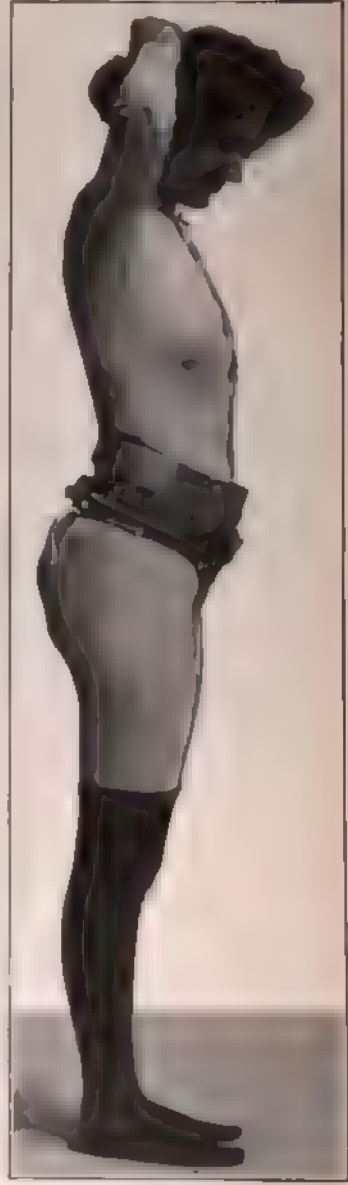
PLATE XXV

FIG. 1



Abdominal Bandage
Properly Adjusted.

FIG. 2



Abdominal Bandage Im-
properly Adjusted.

Measurements for the author's bandage must be exact and should be taken with all the clothing removed (Fig. 98). *J*, carry tape around hips at pubis, ——— inches; *K*, carry tape around hips



FIG. 97.—Author's abdominal bandage, showing construction in detail.

at anterior superior spine of ilium, — — — inches; *L*, carry tape around abdomen six inches above pubis, — — — inches; *M*, width of pelvis between the anterior superior spines of the ilium, with the abdomen well compressed, — — — inches.

An appliance very much in vogue is an adhesive-plaster bandage, by Rose, of New York. It consists of zinc oxide moleskin adhesive plaster one yard long and eight inches wide. From this a pattern is cut as shown in Fig. 99. I am in the habit of placing the patient in the Trendelenburg position in order to apply this plaster bandage. All hairy portions of the body covered by the bandage should be shaved to facilitate removal. The plaster should not include the crest of the ilium, but should run closely along and above it. The epigastric region remains uncovered (Fig. 100). Most patients find this bandage fairly comfortable. Patients are able to bathe regularly while wearing it. They often learn to apply it properly themselves. Unfortunately, it is impossible to keep it in place longer than three or four weeks, owing to the fact that the plaster loses its adhesiveness.

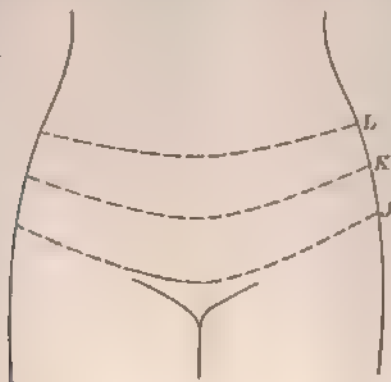


FIG. 98.—Diagram, showing lines of measurements for bandage.

The removal of adhesive plaster from the skin of a patient is accompanied by considerable pain and discomfort. Oil of wintergreen completely destroys the adhesive elements in a very short time. It is not necessary to use more than a small amount of the oil, which is applied directly to the plaster and easily spreads itself throughout the adhesive material. When extensive areas of plaster are to be removed the application of an ointment of *adeps laue hydrosus*, with 10 per cent. of oil of wintergreen incorporated, is even more useful than the oil alone. A little gasoline in a medicine dropper allowed to drop under the plaster so relaxes the adhesiveness that the plaster can be easily removed. Better still, soak the plaster with gasoline applied with a piece of gauze or cotton. The plaster can be thus easily loosened and removed.

CORSETS.—The corsets most in use are those devised by Bardenheuer, Landau, Gallant, and Fitz. Gallant advocates the semi-opisthotonos posture (Fig. 101) as the proper one for the patient

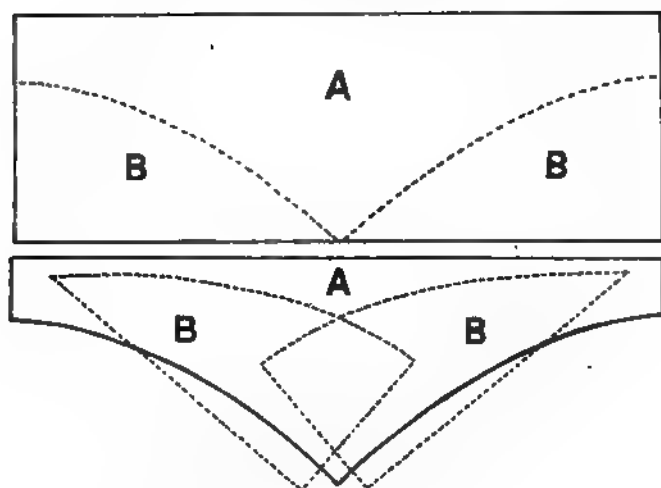


FIG. 99.—Pattern for adhesive belt. (Rose.)

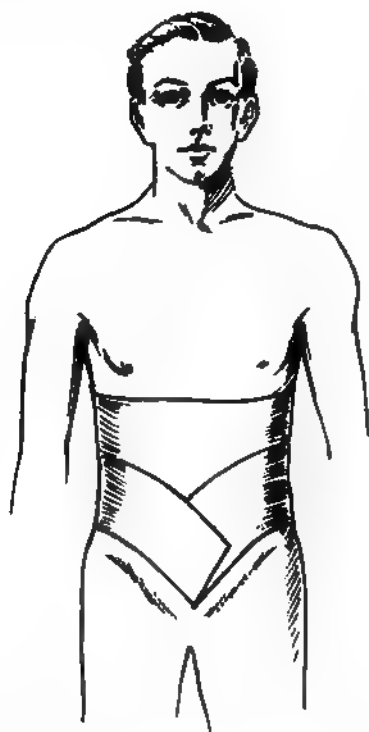


FIG. 100.—Adhesive-plaster belt adjusted. (Rose.)

to assume in putting on his corset. A good corset should be high and snug-fitting in the back, low and loose over the epigastrium, long and close-fitting at the pubes, short behind and well molded to the sacrum from which it takes support.

The corset I prescribe is an ordinary long-hip, straight-front corset, and may be purchased at any up-to-date corset shop. Its main feature is the fact that it laces in front. When properly adjusted it forms a valuable therapeutic factor in the treatment of gastroenteroptosis. This is especially true when the abdomen is prominent, protruding anteriorly to the spines of the ilium. The corset is not so beneficial when the abdomen is flat; in such cases the author applies his abdominal bandage.



FIG. 101.—Position for adjusting corset. (Gallant.)

Method of Adjusting Corset. The corset should be opened the full length of the strings before hooking. After the corset is hooked in front it should be pulled down as far as possible by grasping the lower edge with one hand, the undergarment being pulled up with the other. When the lower edge of the corset is half-way over the symphysis, the garters should be fastened all around, and the buckles so adjusted as to tighten the garters. The corset should be laced from below up to the eighth eyelet like a shoe, thus gradually raising the displaced organs. Now start at the top and lace down to the waist line, leaving the corset loose enough to relieve pressure in the epigastrium. The laces should be tied at the waist line, when the corset will be found to be in proper position. This corset presses over the hypogastrium, so that when it is laced the lower abdomen has become less prominent (Plate XXVI).

PLATE XXVI



Corset Adjusted Correctly.
Side View.

Pregnancy has frequently had the effect of so raising the abdominal organs as to bring about recovery in cases of gastroenteroptosis. Women with ptosis who become pregnant have an increased intra-abdominal pressure, which will vary directly as the uterus increases in volume. Pregnancy produces a marked improvement in the digestive functions in these cases, and there is no reason why this improvement may not be made permanent through proper treatment. It becomes markedly apparent during the later months of pregnancy. Normal pregnancy, then, does not exert any bad influence upon gastroenteroptosis. If after delivery the viscera are sustained with a properly fitted bandage for some time, a contraction of the abdominal walls takes place and in due time the organs will continue in their proper position.

Medicinal Treatment.—Most patients suffering from gastroenteroptosis require iron in some form. They cannot take it internally, owing to its irritating effect upon the gastric mucous membrane. This difficulty may be overcome by hypodermic medication. Of all the iron preparations, ferric citrate has been found best for hypodermic use; as a reconstructive hematinic it is probably the best form of the metal. Combined with arsenic, iron acts as an alterative, and the compound may be used in all cases of cachexia and in all anemias. Iron, arsenic and strychnin in the form of glycerophosphates are indicated in disturbances of a nervous nature, particularly in those neuromuscular cases where there is a marked deficiency of phosphates. The cacodylate of iron is particularly valuable in combating the graver forms of anemia which are found sometimes associated with gastroenteroptosis. Owing to the fact that this preparation is well borne by the kidneys, it can be prescribed in all cases of anemia and kidney involvement.

Of all methods of administering drugs, the hypodermic is giving the best results. The colloidal metals have a pronounced biologic influence on the body. The electro-colloids of iron, silver, gold, copper and mercury can be given intravenously. The cacodylate of iron and the cacodylate of sodium have proved of great value when given intravenously. The Italians have done much to develop this mode of administering pharmacopœial preparations. These preparations are put up in the form of aseptic solutions in hermetically sealed glass ampoules.¹ Their method of administration is as follows: The stem of the ampoule in which the dose is contained is broken off, and the dose drawn up into the syringe under aseptic conditions. The injections are made deep into the gluteal region; they should be given intramuscularly. Iron cacodylate may be safely given intravenously. The following combinations can be used:

¹ By Molteni & Company of Florence, Italy; imported by L. A. Saltzer, of Detroit, Mich.

	Grams
Iron cacodylate	0 03
Iron citrate (green)	0 05
Sodium arsenate	0 001
Iron citrate (green)	0 05
Iron citrate (green)	0 05
Strychnin sulphate	0 001
Iron citrate (green)	0 05
Sodium arsenate	0 001
Strychnin sulphate	0 0005
Iron citrate (green)	0 05
Sodium glycerophosphate	0 05
Sodium arsenate	0 001
Iron citrate (green)	0 05
Sodium glycerophosphate	0 05
Strychnin sulphate	0 001
Iron citrate	0 05
Sodium glycerophosphate	0 05
Sodium methylarsenate	0 10
Strychnin sulphate	0 001
Iron citrate	0 10
Sodium glycerophosphate	0 50
Sodium methylarsenate	0 10
Strychnin sulphate	0 001
Iron citrate	0 05
Sodium glycerophosphate	0 20
Sodium methylarsenate	0 10
Sodium formate	0 15
Strychnin sulphate	0 001

Liquid petrolatum (purified mineral oil) is indicated when desire to lubricate the whole gastro-intestinal tract to facilitate the passage of its contents. The feces are softened and under microscope are found to contain minute globules of the oil. heavy an oil should not be used, for this fails to permeate the material, a desideratum as important as the lubrication of intestinal wall. The oil is not absorbed from the alimentary tract and even in large doses has no poisonous effect. It is useful only as a lubricant, but also as a means of healing superficial lesions of the mucous membrane. It may be given for any irritation of the mucous membrane of the gastro-intestinal tract with absolute safety. The oil inhibits bacterial growth. In tablespoonful doses three times daily it acts as a mild laxative (see page 664).

Improvement in the appetite sometimes follows the administration of a stomachic. Where atony is present the medicinal agents appropriate for that condition may be given (see Chapter XXIV).

Mineral waters, when they seem to be indicated, should be prescribed tentatively according to the principles given for

treatment of atony. In cases where there is much distress, mineral waters are contra-indicated.

Surgical Treatment.—Operative measures have been employed for the relief of severe symptoms incident to gastroenteroptosis of marked degree. One of these is the fixation of the stomach to the parietal peritoneum by the shortening of the ligaments (gastropexy). The operation for nephropexy also has been employed. The lesser omentum of the stomach has been attached to the anterior abdominal wall. The fasciæ of the gastric muscles have been resected and sutured. The colon, hepatic and splenic flexures have been attached to the anterior abdominal wall. A hammock has been made of the great omentum for the suspension of the stomach. Surgical plication of the gastrohepatic and gastrophrenic ligaments with three rows of sutures has been done. The recti muscles have been so united with kangaroo-tendon sutures as to cause overlapping. Many other original surgical measures have been employed. The results obtained by these operations have not, however, been encouraging; and since surgical intervention has proved ineffectual in the treatment of gastroenteroptosis, gastroenterologists have practically ceased to advise it. Better reports on the surgical relief of chronic intestinal stasis by the removal of pericolic membranes, bands, kinks, etc., have been made. (See Chapter XXXIX.)

CHAPTER XXXI.

DISEASES OF THE LIVER.

HEPATITIS; ABSCESS; YELLOW ATROPHY; FEBRILE ICTERUS;
HYPEREMIA; CIRRHOSIS; ATROPHY; SYPHILIS; TUBERCULOSIS;
NEOPLASMS; PARASITES; FATTY LIVER; NEURALGIA.

ACUTE AFFECTIONS OF THE LIVER.

Acute Inflammation of the Liver.—The question whether there is a pure form of primary hepatic inflammation is not easily decided, because in every case of inflamed liver there is a possibility of its having been caused by some other hepatic affection accompanied by inflammatory manifestations. Cases of apparent primary hepatitis occur mostly in the tropics, notably following malaria and dysentery, but also after exposure, dietary errors, or abuse of alcohol. According to the general description, it manifests itself without premonitory signs, with chill, high fever, and pain, and disappears when the patients conform to a correct mode of living. There may, however, be relapses. The etiology of these manifestations is not established, but intestinal toxemia or infection should be considered. Possibly hyperemia of the liver, common in the tropics as a result of dietetic errors and excesses, supplies a groundwork for acute inflammations.

In northern countries hepatic inflammation is characterized by a more or less pronounced swelling of the liver, pain, tension and pressure in that organ, sometimes by a slight rise in temperature, and sometimes icterus; but all these manifestations recede in a short time. As already stated, it is difficult to decide whether the inflammatory state of the liver which they express is or is not secondary to some other hepatic disorder.

Treatment.—The treatment consists of rest in bed, abstinence from alcohol and all injurious articles of diet, and the use of light, non-irritating foods. In the first stage of the illness mild laxatives are in order, such as calomel 0.03 Gm. ($\frac{1}{2}$ grain) one or two doses, then rhubarb, cascara, senna, etc. After a cure has been effected alcohol should be avoided for a long time, and the diet should continue bland until all danger of relapse is past. Should there be frequent relapses, a change of climate is desirable.

Abscess of the Liver.—Suppurative hepatitis is either primary or secondary. Primary abscess of the liver is usually of traumatic origin, occurring after contusions or direct injuries from stabs or

shots. Secondary abscess is due to invasion of the liver by bacteria and parasites from other parts of the body. Septic processes are of first importance, the infectious material spreading to the liver in the form of emboli, especially in cases of ulcerous endocarditis. Other sources of infection are ulceration and gangrene of the lungs, bronchiectasis, putrid bronchitis, all kinds of ulcers and abscesses occurring from birth to old age, especially appendicitis, salpingitis, suppurative inflammation of the gall bladder and biliary ducts, and finally, in the tropics, dysentery, in which the *Endamebæ coli* migrate into the liver. Although the possibilities of the occurrence of liver abscess are almost unlimited, it develops comparatively rarely in the temperate zones, both sexes being affected in about the same proportion. In the tropics, however, males are oftener attacked, evidently as a result of the abuse of alcohol and immoderate eating and drinking.

According to the size and number of the abscesses, the liver is more or less enlarged, either the whole organ or only one of the lobes being involved. When the abscesses contain large quantities of pus they often push out the overlying skin, forming palpable protuberances, while superficial abscesses which are not subjected to high pus pressure form fluctuating sacs. The tropical abscess has a predilection for the right lobe and is usually solitary. The pus is either of a creamy consistency, or more serous, biliary or bloody, of chocolate color, often of putrid odor, and may contain numerous tissue threads; at times it contains small gallstones. The abscesses are mostly encapsulated in connective tissue, which is apt to become hard and thick, lined with a villous stratum of changed hepatic tissue, pus and bacteria. The neighboring hepatic cells show fatty degeneration, occasionally gangrene and necrosis, with thrombosis and thrombophlebitis of the hepatic veins. The suppurative inflammation frequently spreads beyond the liver, causing adhesions and encapsulated abscesses which may be subphrenic or located in the pleura or the lung. Perforation into the abdominal cavity, stomach, intestine or the larger vessels occurs less often. Nevertheless, perforation into the renal pelvis, esophagus and pericardium have been reported. Even descending abscesses along the ligamentum teres, with perforation of the abscess at the umbilicus, may occur.

Symptoms.—Most secondary abscesses show but indifferent symptoms, or none at all, and are usually completely concealed by the underlying affection. They are often not discovered until the cause (appendicitis) has run its course. A symptom of abscess is high temperature of a remittent or intermittent character, complicated by chills. Considerable night-sweats are not infrequent. On the other hand, fever may be entirely absent in many cases, especially in the later stages. Changes in the shape of the liver may clinically be observed by inspection and palpation,

according to the size and number of the abscesses. When the latter are fresh, there is pain, both spontaneously and upon pressure; the pains may radiate toward the back, right shoulder and right upper arm. The skin generally has a sallow color. There is considerable emaciation. The spleen is often enlarged and the digestion disturbed. Urobilin and urobilinogen have been found in the urine. Involvement of the lungs and pleura or reflex irritation of the phrenic nerve may cause considerable paroxysms of coughing. High position of the diaphragm may give rise to dyspnea. The diagnosis is made, if possible, by test puncture. The differential diagnosis between empyema of the pleura, subphrenic abscess, and liver abscess, can usually be correctly made, especially with the aid of the Roentgen ray, if all the existing possibilities are duly taken into consideration.

With the patient in a semiprone position, traction on the umbilicus in the direction of the pubis will often elicit pain in the region of the liver. This referred pain is present in cases of cholecystitis, hepatic abscess, and other diseased conditions of the liver.

Spontaneous resolution, or spontaneous perforation of the pus outward, followed by a cure, is always exceptional. The disease runs a fatal course unless the pus can be evacuated.

Treatment.—The only rational therapy is surgical. When the presence of an abscess has been established by puncture, a broad incision is made at the point of puncture. If, in the absence of pus after a test puncture, there are other symptoms pointing to the probability of an abscess, the latter may sometimes be found by palpation and opened after laparotomy or costal resection. The abscess cavity should be irrigated with saline solution.

If the abscess cannot be located by puncture, internal treatment is in order. The latter can, of course, be symptomatic only. Pain and swelling of the liver are relieved by cold compresses, ice or leeches applied to the hepatic region. The intestinal function is incited by mild laxatives and bitters. The diet should be light, pulpy, fluid and non-irritating, but withal nutritious and concentrated, in order to provide for maintenance of the strength necessary for the operation which may have to follow later. Dysentery, if present, should be carefully treated (see Chapter XLIII). Tropical patients had best be sent home.

Emetin hydrochlorid has proved curative in amebic abscess of the liver, injected after withdrawal by trocar of the pus, and later subcutaneously (see page 723).

Acute Yellow Atrophy of the Liver.—Acute parenchymatous hepatitis, or icterus gravis, is relatively rare and occurs after infectious diseases, typhoid, recurrent fever, erysipelas, diphtheria, in the early stages of syphilis, and particularly during pregnancy and the puerperium. Accordingly, the female sex is oftener attacked than the male. The early symptoms are hardly noted—slight dyspepsia

and a little jaundice; but after these have persisted for one or two weeks the case suddenly assumes a serious form, with vomiting, eructations, nausea, and pain in the back and in the hepatic region. The patient becomes listless and a prey to hallucinations and delirium, with increasing icterus of the gravest form, death resulting in a few days. During this time the liver decreases in volume, and the hepatic dulness may sometimes (though not always) completely disappear, while the spleen becomes enlarged. There is considerable rise in temperature toward the end. Sometimes there are cutaneous hemorrhages and epistaxis. The quantity of urine is considerably decreased, and, aside from biliary pigment, albumin and casts, the urine contains the aminoacids leucin and tyrosin, which are characteristic of the disease; also sarcolactic acid, oxyhydrocyanic acid, and peptoid bodies. Leucin and tyrosin have also been found in the gall bladder. At autopsy the liver is found to be shrunk to one-half of its size or less, the left lobe being more involved than the right; the whole organ shows a dirty reddish-yellow discoloration; its surface is often spotted, brownish-red and gray areas alternating with yellow and greenish-yellow. The cut surface is also discolored, the yellow parts being more prominent than the red. Under the microscope extensive degeneration of the hepatic cells is seen, together with a small-celled interstitial proliferation. Leucin and tyrosin may be found in the interior of the hepatic and portal veins.

This is a primary acute affection of the liver, possibly due to a kind of toxemia from the intestine through the portal vein. Specific microorganisms have not been found. The affection terminates fatally, the number of reported apparently spontaneous cures being exceedingly few.

Treatment. The treatment can only be symptomatic, consisting of ice and morphin for vomiting and pain, ice-bags and cold compresses applied to the liver and head. The vitality of the patient should be kept up by light nutrition, so far as possible.

Febrilis Icterus.—This acute febrile jaundice is called "Weil's disease" because it was first described by Weil in 1866. It usually occurs in young men, commencing with chills, high fever, anorexia, thirst, and vomiting. The temperature falls between the third and the fifth day, then rises, and again lytically declines to the middle of the second week. Jaundice usually occurs during the first days or when the fever declines; sometimes, however, the feces are not discolored and the urine is free from bilirubin, suggesting hemolytic icterus. The fever is accompanied by painful swelling of the liver, enlargement of the spleen, acute nephritis, muscular pain, especially in the calves, dyspeptic symptoms, and manifestations of excitation. Exanthemata, herpes, hemorrhages into the mucous membranes and retinal hemorrhages have also been observed. The affection usually takes a favorable course.

The few cases which have come to autopsy have not shown any characteristic hepatic changes.

A specific cause of the disease is now supposed to be a plicate spirochete (Uhlenbuth and Fromme) which does not coil in regular spirals. This parasite shows swellings and nodules and is called *Spirocheta nodosa*. Japanese investigators have inoculated guinea-pigs with the blood of patients suffering from infectious hemorrhagic jaundice resembling Weil's disease and thus transmitted the disease with all its characteristic symptoms. They then discovered, in the blood of the animals as well as in that of the human patients, a microorganism which they designated *Spirocheta inter-hemorrhagica*. This organism has been demonstrated to be the cause of Weil's disease. It is now believed that the strains of these spirochetes are identical. The spirochetes are found constantly in the blood. Spirochetal jaundice has shown a mortality of 10 per cent. on both the eastern and western coasts. The organisms are extremely motile and can penetrate the unbroken skin. Noguchi has found them in wild rats in New York City. The disease is supposed to be spread by rats. In many cases, apparently, the icterus has followed ingestion of decomposed food, especially meat; but toxemia also may be responsible.

Treatment.—The treatment consists in the injection of serum taken from a convalescent horse. Good results have followed this therapy. The fever is treated with antipyretics (quinin, pyramidon); the dyspeptic manifestations are relieved by mild laxatives, the manifestations of collapse by analeptics, and the nervous excitement by narcotics. Among the latter may be recommended subcutaneous injections of morphin-scopolamin (scopolamin hydrobromid 0.0005, morphin 0.01); also 2 or 3 grams (gr. xxx xlv) of chloral hydrate per rectum. Tepid baths, gradually cooled down, are advisable. In some cases the intravenous injection of arsphenamine or neocarsphenamine is of great benefit (see page 534).

CHRONIC AFFECTIONS OF THE LIVER.

Active Hyperemia.—This hyperemia is due to an excessive supply of arterial blood, caused by persistent dietary errors (too abundant and too frequent meals); alcoholism; overindulgence in coffee, spices and meat; infectious diseases (malarial, typhoid or yellow fever); climatic injuries (tropical climate); menstrual irregularities; trauma in the hepatic region; or biliary colic. All these may cause acute and chronic hyperemia. The liver is enlarged, a condition which is particularly characteristic when the enlargement is subject to frequent changes or to complete temporary disappearance. At the same time the organ is sensitive to pressure, even pressure of the clothing. The pain is occasioned by pulling and pressure on the hepatic capsule, which contains numerous

sensory nerves. There is a sensation of heaviness in the gastric and hepatic regions; the patients feel a subjective disturbance of digestion, though the digestive organs are intact; they feel very nervous and irritable, which is accounted for by the persistent recurrence of the causative noxious factors and consequent hyperemic condition.

Diagnosis.—The diagnosis of active hyperemia is not always easy and can only be verified by continued observation. The hyperemia may be the first sign of a beginning grave hepatic affection. On percussion the lower edge of the liver is extremely painful.

Treatment.—In the first place the causative factors are to be eliminated—which means, primarily, restriction in the quantity and quality of food. Only small portions of meat, preferably very tender and soft, should be allowed. A lactovegetable diet, consisting of milk, farinaceous food, buttermilk, yoghurt, green vegetables, legumes, fruit, and tea, is likewise in order; while heavy, very fat meats, smoked and pickled meats, alcohol and strong condiments are excluded. The intestinal function is to be gently excited by the vegetable bitters or alkaline mineral waters, or mild vegetable laxatives. In chronic conditions a stay at West Baden, Saratoga, Kissingen, Carlsbad, Betrich or Vichy is to be considered, always provided the patient is willing to adapt his entire way of living to the requirements of the cure. Local pains are relieved by cold compresses, ice-bags, dry cupping, or deferent plasters.

Passive Hyperemia.—Congestion of the liver is met with in all affections which impede the flow of the venous blood toward the heart. This, therefore, includes all valvular irregularities and other disorders of the heart and large vessels which lead to cardiac weakness. Again, all affections of the lungs or other pectoral organs, especially also kyphosis and scoliosis, lead to engorgement of the liver. In these conditions the liver is hypertrophied, dark blue to red in color, and of hard consistency. The lobular centers in the transverse section are more vividly red than the periphery, which accounts for the characteristic appearance of the cut surface designated as nutmeg liver. In persistent engorgement, parts of the liver undergo atrophic obliteration, causing proliferation of the intralobular connective tissue, which means shrinking of certain parts of the liver, while other parts will correspondingly protrude. This causes the liver to become harder and smaller. The surface is more granulated, showing a certain similarity to cirrhosis and constituting the atrophic nutmeg liver.

Symptoms.—The hepatic congestion is accompanied by a feeling of pressure and pain in the affected region. Usually, however, these symptoms are less pronounced than those which are caused by the underlying affection. The diagnosis is established by inspection, palpation, and percussion.

Treatment.—The treatment is directed to the underlying affection. So far as the liver is concerned, light and not abundant nutrition, attention to the fecal evacuation, and avoidance of the injurious influences mentioned in the treatment of active hyperemia, are of importance.

Atrophic Cirrhosis.—Chronic interstitial hepatitis, or atrophic cirrhosis, is a disease of the liver with marked increase of the connective tissue, which afterward contracts, producing atrophy and degeneration and giving the organ a granular, yellow appearance (granular atrophy) due to the coloring of the acini by the biliary pigments.

Etiology.—Atrophic cirrhosis, known as Laennec's disease, may be due to any one of a number of causes. Although there is no doubt that the rôle of alcohol as a causative factor has been overestimated, the fact remains that a large proportion of cases are to be attributed to the abuse of alcohol. Many authors assume an endogenous development of hepatic cirrhosis. The endogenous theory, if accepted, accounts for pancreatic cirrhosis and similar injuries to other organs (as the spleen), as well as for hepatic cirrhosis. It is also assumed that toxins from chronic gastric and intestinal catarrh are conducted to the liver through the portal vein, there causing the characteristic changes of the periportal tissue. Alcohol thus comes in for a share of the blame on the ground that its use, or abuse, favors the development of gastric and intestinal catarrh. It has been experimentally demonstrated, though not in human pathology, that lead, organic acids and other substances may produce cirrhosis. Nor is there any doubt that infectious diseases predispose to cirrhosis, as has been observed in syphilis, tuberculosis, and malaria. As a matter of fact any disorder capable of setting up acute hepatitis can lead to cirrhosis, because hepatitis may cause cirrhosis—for instance, after scarlet fever. Japanese authors have reported that hepatic cirrhosis has occurred after infection with *Schistosoma japonica*, an intestinal parasite, probably by direct mechanical irritation of the hepatic tissue. So far as the great majority of cases of atrophic cirrhosis of the liver are concerned, it must be assumed in a general way that they are caused by a toxin introduced by the portal vein. It has been reported that atrophic cirrhosis has occurred in gout and diabetes. Individuals between the ages of forty and sixty are the most frequent victims, while in the very old and the very young the affection has been found but rarely.

Pathology.—The pathologic changes of the liver in cirrhosis are characteristic. As a rule the shape of the organ is unaltered, but it is frequently so shrunken in size as to have lost half its weight. Its surface is covered with small excrescences, from pin-head to pea size, a smooth surface being the exception. The hepatic capsule is often cloudy and thickened in places by callosi-

ties. The liver substance is very hard, so hard that it can only with difficulty be cut. In most cases the left lobe is more involved than the right. The cut surface exposes the proliferated connective tissue in the shape of a whitish, reddish or gray net, the meshes of which contain what has been preserved of the hepatic parenchyma. Microscopically, these coarse anatomic changes are seen to have been caused by a rather advanced degree of proliferation of the interlobular connective tissue. The latter, encircling the lobes, sends out solitary strands between the various acini. The development of the connective tissue, however, is always interlobular. The liver cells, which exhibit cloudy swelling and fatty degeneration, gradually perish in the zone of contact with the connective tissue. There are inflammatory changes of the blood-vessels in the form of periphlebitis or phlebitis with partial obliteration of the veins. Parts of the connective tissue, too, show small-celled infiltration. At the same time neoplasms of the hepatic tissue and particularly of the biliary capillaries are occasionally found.

Symptoms.—The early symptoms of hepatic cirrhosis are dyspeptic, with eructations, constipation or diarrhea, and a sensation of weakness. As these symptoms increase, the face becomes sallow and the sclerae painful and discolored. These symptoms may persist for years, in varying intensity, before the unmistakably characteristic manifestations of cirrhosis appear. Gradually, changes in the liver itself become apparent—enlargement followed by shrinking, or conspicuous hardness. Unless the abdominal walls are very thick, unevenness of the hepatic surface can be discovered by palpation. In most cases there is little or no sensitiveness to pressure. It is only with the onset of inflammation of the hepatic capsule that any marked degree of pain is experienced. In a considerable number, but by no means all cases, there is gradual enlargement of the spleen; it is probable that this splenic hypertrophy is due to the same toxins that affect the liver; in most cases there are no characteristic changes in the structure of the spleen. Jaundice occurs in some cases, but not often. The discoloration of the skin, referred to above, is, however, characteristic. The feces are colored accordingly. Defecation is often irregular, diarrhea alternating with constipation in the presence of pronounced meteorism. One of the characteristic symptoms is abdominal ascites, which develops *pari passu* with the hindrance to the portal circulation caused by connective-tissue proliferation and shrinking of the hepatic lobes. But portal stasis is surely not always the cause of ascites; it is probable that chronic inflammatory changes in the hepatic capsule and the peritoneum, together with contraction of the mesentery, favor its occurrence. In the initial stage of ascites the swelling abdomen, pushing upward on

the well-filled stomach, causes elevation of the diaphragm, displacement of the heart, and compression of the lungs. The ascitic fluid is usually limpid and yellowish, and its specific gravity goes up to 1015; the albuminous content amounts to 0.5 to 2 per cent. As the damming of the hepatic circulation increases, the portal blood endeavors to find other means of reaching the vena cava, and the possibility of its doing so through the gastric and esophageal veins is to be considered. Owing to the plethora of these veins and the portal stasis, varices are easily formed which may burst in the esophagus, giving rise to hemorrhages of varying extent. Similarly, overdistention of the hemorrhoidal veins may cause hemorrhoids and hemorrhages. Should, then, the mesenteric and epigastric veins anastomose, the veins, notably of the anterior abdominal wall, protrude in the shape of bluish cords, especially in the umbilical region, forming the so-called *caput medusæ*. Again, new venous tracts in the ligamentum teres may run to the veins of the abdominal walls. In a small number of cases of atrophic cirrhosis there is no ascites. Fever is rare. The quantity of voided urine is small in proportion to the collection of ascites. Unless icterus is present, there is no demonstrable biliary pigment, but there are urobilin and urobilinogen. In most cases patients become anemic to a rather marked degree. There is no difficulty in making the diagnosis in fully developed cases, but in the initial stages the anamnesis is the principal reliance of the diagnostician. The diminished content of fibrinogen in the blood is an aid in the diagnosis (see page 384).

On account of the destruction of its cells the liver does not function normally. This can often be ascertained by the several functional tests:

Tests for Lipase.—Normally the blood contains a definite percentage of lipase or lipolytic ferment. That one of the functions of the liver is to control within normal limits the circulation of lipase in the blood is inferred from the fact that in certain diseases of the liver the percentage is increased. It was found by Whipple, in collaboration with Mason and Peightol, that after acute hepatic injury from chloroform the percentage of lipase in the blood serum or plasma was always increased. For estimating the amount of lipase in the blood, 1 Cc. of blood serum is placed in each of four tubes, and 4 Cc. of distilled water added to each. Two of these filled tubes are used as controls. To each of the others 0.26 Cc. of ethyl butyrate (butyric ether) is added. All four are shaken, corked, and incubated at 38° C. for eighteen to twenty-four hours, then cooled in ice-water, 3 drops of azolitmin added to each, and titrated in pairs with decinormal acid and alkali solutions—the controls with the former, the others with the latter. The controls show the natural alkalinity of the blood; and to this figure is

added the free butyric acid figure found in the lipase test tubes, to determine the total butyric acid formed.¹

Levulose Test. The levulose test for hepatic insufficiency depends upon the finding of unassimilated sugar in the urine. The patient is given 60 Gm. (2 ounces) of levulose on an empty stomach. If a healthy individual ingests this quantity, a positive reaction is almost never obtained. The test is a functional one. The physiologic duty of the liver is to store up glycogen. The liver metabolism is so deranged in certain chronic diseases, such as cirrhosis, atrophic processes, and, in general, destruction of the liver parenchyma, that levulose is found in the urine. For testing in levulosuria, a simple Nylander reaction is sufficient. To 10 parts of the urine under test add 1 part of the Nylander solution, consisting of 2 parts bismuth subnitrate and 4 parts sodium and potassium tartrate dissolved in 100 parts of a 10-per-cent. solution of sodium hydroxid. The presence of glucose is indicated by black coloration or precipitate.

Phthalein Test.—It has been found that phenoltetrachlorphthalein is eliminated entirely by way of the bile. This drug has been utilized in testing the function of the liver, just as the function of the kidneys is tested with sulphonephthalein. A freshly prepared, boiled, isotonic solution of phenoltetrachlorphthalein in distilled water is administered intravenously, and after free purgation the stools are collected during forty-eight hours and tested for the drug. About 30 to 40 per cent. of the phthalein is normally recovered. This test for liver function is a difficult one, and recent reports indicate that the information thus adduced is not as valuable as was at first supposed.

Urobilin.—In the majority of cases sufficient functioning cells persist to enable the liver to maintain its metabolic processes in approximate equilibrium. Urobilin is normally formed from the biliary coloring material in the intestine. This material is carried to the liver and is there transformed into biliary pigment. Should the parenchyma of the liver be unable to properly perform this function, urobilin or urobilinogen is found in the urine. Urobilinuria is the invariable accompaniment of disease of the liver. It is probably the first appreciable symptom of cirrhosis of the liver and the first symptom of beginning obstruction to the outflow of bile, and it is the last to disappear when the bile stasis has been corrected. When obstruction is so complete that no bile enters the intestine, there can be no urobilinuria; hence the absence of urobilinuria in diseases of the liver testifies to complete obstruction of the bile passage.

Prognosis.—Generally speaking, the prognosis is unfavorable. Even though the affection may be protracted for years and remain

¹ Lowenhart, American Journal of Physiology, 1902, vi, 331.

at a standstill for a number of years, it is fatal in the majority of cases. The number of cases in which a final cure, or at least a resolution of the manifestations, has been claimed, is very few.

Treatment.—All injurious indulgences are to be avoided. Alcohol is rigorously prohibited. Attempts should be made to raise the power of resistance by rest, good nursing and diet, fresh air and skin culture, in order to offer the liver a certain degree of participation in the general recuperation. The diet should be light and bland, but plentiful, corresponding to the protective treatment of chronic gastric or intestinal catarrh. Meat should be sparingly partaken of, and salt avoided, to promote diuresis as in cases of nephritis.

Recently Einhorn has recommended duodenal alimentation (see page 500) in hepatic cirrhosis, assuming that it facilitates hepatic function by stimulating the portal circulation.

Frequent baths (with or without medicaments), ablutions, and friction with water or spirituous solutions are indicated. Increased diuresis will induce absorption and elimination of the ascitic fluid. Elimination should be promoted at an early stage by the administration of mild laxatives (senna, rhubarb, cascara sagrada, phenolphthalein). Diuretics, such as theobromin sodiosalicylate, theobromin, theophyllin, euphyllin, to be administered by mouth or as suppositories several times daily, come in for particular consideration. It is advisable to prescribe theophyllin and euphyllin periodically because their effect extends over a few days only. After a lapse of one or two weeks they may again be used for a few days. Caffein sodiosalicylate has an excellent effect. So long as the kidneys are healthy, concentrated solutions of urea (20, aq. ad 200, one tablespoonful every hour) are advantageous in some cases. Calomel in large doses is sometimes used as a last resort in order to avoid abdominal puncture, 0.2 Gm. (3 grains) being given four times daily. It may also be combined with urea according to the following:

	Gm. or Gs.	
R Hydrargyri chloridi mitis	0 1	gr ʒss
Urea pura	1 (0	gr. xv
Misce et ft pulv no xxv.		

Sig.—One powder three times daily.

The administration of calomel, however, demands great care, owing to the grave stomatitis it may occasion, and also because its excretion appears to be hindered by the hepatic affection.

Keratin seems to be an antisclerotic remedy of some value. The patient is given 1 Gm. (15 grains) of keratin three times a day, and the dose is gradually increased up to 2 Gm. (30 grains). It must be given for several months. Should there be a gastro-intestinal irritation, with diarrhea, bismuth salicylate is to be given in doses of 0.5 Gm. (7½ grains) every three or four hours. Fibrolysin has been used in some cases (see page 484).

Should syphilis enter into the question of etiology, iodids may be administered tentatively in large doses in the shape of sodium iodid, potassium iodid, iodipin, or iodalbin. The need of caution in the administration of calomel, referred to above, applies to other mercurials as well.

Eichhorst warmly recommends bitartrate of potassium to relieve ascites. When used for several weeks, this compound is said to be strikingly effective, probably on account of its diuretic properties.

	Gm. or Cc	
R—Decocti radici althææ	10-18 0	℥iiss-℥vss
Potassii bitartratis	15 0	℥iv
Syrupi	20 0	℥v
Misce.		
Sig —One tablespoonful every two hours, well shaken		

Should it be impossible to control the ascites by any means whatever, paracentesis will have to be resorted to. The consensus of opinion favors an early puncture, before very large quantities of fluid have accumulated, even when no more than $1\frac{1}{2}$ to 2 liters is present. Indeed, objections are no longer raised to very frequent abdominal punctures, because frequent and early relief of the abdomen from the ascitic pressure has a very favorable effect upon the local and general circulation and incites the kidneys to greater activity. The question whether all or only part of the fluid should be evacuated is not of very great importance, because there is certain to be reaccumulation after the first puncture, so that a fresh puncture becomes imperative in any case. This procedure is very simple. An ordinary pleural or abdominal trocar is used. With the patient in the sitting posture, the puncture is made immediately above the symphysis in the median line, the bladder having been previously evacuated. When the patient is in the lateral decubitus the puncture is made in the elongated median axillary line. Should intestinal loops present in the opening, they may be pushed aside by a sterile soft-rubber catheter or a mandrin which fits into the trocar, evacuation being hastened by varying the position of the patient or by manual pressure upon the various abdominal regions. The opening is closed with adhesive plaster after the trocar has been removed. In the event of any fluid oozing out, the opening may be sutured, or a needle is thrust into the elevated skin fold and a thread wound around it to compress the puncture.

Surgical Treatment.—Atrophic cirrhosis of the liver has also pressed surgery into its service for the relief of portal stasis. Talma and Drummond suture plethoric abdominal organs (omentum, spleen, gall bladder) to the anterior abdominal wall, embedding the same in a subperitoneal pouch, in the hope that the veins of these organs will communicate with the veins of the abdominal walls and form a collateral circulation. This procedure is particu-

larly suitable for those forms of cirrhosis in which icterus is lacking and those in which the manifestations of engorgement occupy the foreground. These authors report a few cases in which a cure has been effected in this way.

Ech's fistula consists of an artificial communication between the portal vein and the vena cava. Since we have been able to better the technic of uniting bloodvessels, this surgical operation has been more successful than formerly.

Hypertrophic Cirrhosis.—Hypertrophic cirrhosis of the liver, or Hanot's disease, is essentially different from the form just described. The connective-tissue proliferation is located extra- and intra-lobularly and shows no tendency to reduce the size of the liver. The material feature of the affection consists in the fact that the liver cells themselves become enlarged, that their protoplasm and nuclei increase in volume, and also that the number of the nuclei increases. Numerous new biliary capillaries are formed at the same time. These changes of the hepatic tissue cause considerable enlargement of the entire organ which persists during the continuance of the affection. The liver may attain to a weight of $2\frac{1}{2}$ to 4 kilos. It is hard and smooth to the touch. The capsule on examination is often found cloudy and callous, due to perihepatic changes. As a consequence, there are often adhesions to the neighboring organs. Cross-section of the parenchyma shows yellowish green, and the glistening strands of connective tissue can be seen to traverse the parenchyma.

Etiology.—The etiology of this affection is still entirely unexplained. Alcohol does not appear to play any important rôle. Some authors consider infectious cholangitis the cause. The patients are usually men between twenty and thirty years of age, although the disease has been observed in children.

Symptoms. The early symptoms are vaguely dyspeptic, followed by jaundice and hepatic discomfort, a sensation of tension and heaviness in the abdomen, lassitude, and irregular consistency of the stools. Gradually, permanent jaundice develops. The liver becomes enlarged and indurated, a splenic tumor being formed at the same time. Hypertrophic cirrhosis of the liver is characterized by the absence of all signs of portal stasis, especially of ascites. The quantity of urine is abundant. The urine contains biliary pigment and urobilinogen. The reserve force and sustenance of the patient are rapidly impaired, and the skin becomes dry. The jaundice causes considerable itching. The stool is but seldom acholic. At times there is fever. Toward the end, hemorrhages from the nose, gums and skin are common. Patients may, however, continue to live tolerably well for years until the increasing debility renders them unfit for work; and increasing cardiac weakness and general tabescence denote the near approach of death.

Diagnosis.—It is always difficult to make a safe diagnosis. In the initial stage the differential diagnosis between atrophic and hypertrophic cirrhosis is especially difficult. A safe diagnosis, also, as against carcinoma, can usually not be made until a later stage has developed.

Prognosis.—The prognosis is unfavorable.

Treatment. The general treatment coincides with that of atrophic cirrhosis. Einhorn's plan of duodenal alimentation (p. 500) might also be taken into consideration, this author having often observed prompt reduction in the size of the enlarged liver as a result. Iodin preparations may be used tentatively. A number of authors recommend calomel, 0.01 to 0.02 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain) three to five times daily, in the beginning of the treatment, to be given for a week, followed by an interval of several weeks. The possibility of resulting stomatitis should never be lost sight of.

Hypertrophic Cirrhosis of the Liver in Bronze Diabetes.—This affection is characterized by the well-known triad of abnormalities, diabetes mellitus, pigment deposits in the skin and internal organs, and hypertrophic cirrhosis of the liver. It is of particularly frequent occurrence in England and France, and but rarely observed elsewhere. The conspicuous symptom is the brown or dark gray discoloration of the skin, which, however, does not, as in Addison's disease, extend to the mucous membrane. At the same time the symptoms of moderate or grave diabetes are present, as well as the symptoms of hepatic cirrhosis. Anatomically we find the hypertrophic form of hepatic cirrhosis, plus abundant deposits of brown pigment in the liver cells and connective tissue. This is iron pigment, and the liver is consequently very ferruginous. This pigment is also found in other organs, such as the pancreas and spleen. It is found in the cutis rather than in the epidermis. Jaundice is absent in this form of cirrhosis.

Treatment.—Here, again, the treatment is simply symptomatic, chiefly intended to relieve the diabetes and cirrhosis.

Biliary Cirrhosis.—Biliary cirrhosis is a form of Laennec's cirrhosis which has developed from mechanical occlusion of the biliary excretory ducts, especially the ductus choledochus, or is due to calculi, tumor adhesions, or scars. However, every persistent occlusion of the choledochus in man does not necessarily lead to cirrhosis.

At first the liver is enlarged, firm and smooth. Transverse section shows an intensely greenish-yellow color, due to the widening of the biliary passages and the diffusion of the biliary pigment. The hepatic cells degenerate under the influence of the engorged bile. At the same time there is considerable tissue proliferation, partly in the neighborhood of the biliary ducts, partly between the lobes, which gradually increase in size, finally assuming an anatomic picture identical with that of Laennec's cirrhosis.

This form of cirrhosis is attended at the very beginning by con-

siderable jaundice. Unless the biliary stasis is relieved, a splenic enlargement will develop, together with ascites, leading to a fatal termination in a comparatively short time as a result of cholemia and circulatory disturbances. The diagnosis of cirrhosis when the ductus choledochus is occluded cannot, as a rule, be made until after death.

Treatment.—The treatment is symptomatic only and coincides with that of atrophic cirrhosis, unless surgical intervention be invoked.

Cirrhosis Occurring in the Course of Affections of the Circulatory Organs.—Cirrhosis may develop from passive hyperemia of the liver in arteriosclerosis, cardiac disease, and portal thrombosis.

Pericarditic pseudohepatic cirrhosis, first described by Friedel Pick, does not correspond to the nature of cirrhosis proper. Chronic pericarditis, with adhesions between the pericardial layers, is very often accompanied by chronic hepatitis with hyperplasia of the liver. The capsule of the liver is often exceedingly thickened and may have a perfectly white appearance; hence Curschmann's "sugar-coated liver" ("Zuckergussleber").

ATROPHY OF THE LIVER.

Brown Atrophy.—General disturbances of nutrition, senile marasmus, inanition and cachexia affect the liver as well as other organs; it becomes smaller, darker, browner, tough and coarse to the touch, and has a wrinkled capsule. The liver cells, too, are atrophied, contracted, and filled with dark pigment. Hence the term "brown atrophy." This form of atrophy may also be caused by an isolated disturbance of hepatic nutrition—for instance, by involvement or compression of the portal vein.

Red Atrophy. Persistent venous congestion of the liver may also lead to atrophy of the parenchyma, which is called "red" from the fact that the liver assumes a bluish-red color, setting off the whitish network of the acini sharply, especially as seen on cross-section (cyanotic nutmeg liver). The affection is caused by pressure of the plethoric central veins upon the surrounding cells, which gradually atrophy. The clinical manifestations correspond to those of passive hyperemia of the liver; occasionally light icterus accompanies the other symptoms.

Treatment has to be restricted to the underlying cardiac and pulmonary affections.

Partial Atrophy.—This form of atrophy is caused by compression of isolated parts of the liver by tumors or other processes, intra- or extrahepatic. In this way obliteration of the cells and vessels may occur, together with a circumscribed inflammation, thickening and cicatrization.

SYPHILIS OF THE LIVER.

Congenital Syphilis.—This form of syphilis is already present in the new-born or develops during the first months of life. Infants with congenital syphilis of the liver are emaciated, ill-nourished, and stunted. The abdomen is usually distended. The liver is enlarged, smooth and indurated. The spleen also is apt to be enlarged. Other signs of congenital syphilis are frequent, such as glandular swellings, ozena, pemphigus, etc. Pathologically, the syphilitic liver of the new-born is distinguished by proliferation of the interstitial connective tissue, starting from the capsule and spreading interlobularly in the course of the disease. In most cases of congenital hepatic syphilis there is also perihepatitis with adhesions to the neighboring organs. Gummata are rather rare and occur only in the shape of small nodules. Owing to the connective-tissue proliferation, congestive manifestations and ascites may develop, as in atrophic cirrhosis, and the liver may also undergo atrophy. In the new-born and infants, jaundice is likely to be present. The little patients, becoming more and more debilitated from week to week, contract gastric and intestinal disturbances which lead to early death.

Acquired Syphilis.—Acquired syphilis produces characteristic changes in the liver. Here, again, there is considerable proliferation of the interstitial connective tissue, with hypertrophy or atrophy of the organ. Gummata are not uncommon—from the size of a pea to that of an orange; they are situated usually near the surface of the liver, often immediately under the capsule. These gummata are firm-tissued, but readily break down from interior necrosis, gradually undergoing cicatrization and forming coarse scar tissue, the shrinking of which may grotesquely alter the shape of the liver and its capsule. This process is usually accompanied by pronounced perihepatitis and adhesions of the liver to neighboring organs. Small, diffuse, inflammatory foci are of rarer occurrence. The gummata may also cause fatty degeneration and atrophy of the hepatic parenchyma.

Symptoms. In adults syphilis of the liver may persist for a long time without giving rise to any marked symptoms. In the course of time pains will develop in the hepatic region; these are usually due to enlargement of the liver or friction upon contact with perihepatic processes. A striking and characteristic feature of many cases consists of intermittent fever and chills. In the absence of other etiologic factors, fever and hepatic changes are always suggestive of syphilis. The hepatic pains are sometimes very severe, occur in paroxysms, and are particularly aggressive during the night. The spleen, in some cases, is enlarged. Jaundice is not a constant symptom. Ascites often appears at an early stage.

Diagnosis.—The diagnosis of syphilis of the liver is usually very simple in the new-born. In older children and in adults, where acquired syphilis may have to be considered, the anamnesis is of importance, likewise the Wassermann or Noguchi reaction and the result of antisyphilitic treatment. The prognosis is not unfavorable, as a rule. Under appropriate treatment a cure is often effected, or at least resolution and arrest of further progress. On the other hand, some cases are very resistant to treatment.

Treatment.—The treatment is antisyphilitic. Iodin, mercury and arsenamine suggest themselves (see page 534). Extensive gummatous formations have been subjected to surgical treatment, and gummatous nodules have been successfully enucleated. When, in dubious cases, antisyphilitic treatment is not successful, an exploratory laparotomy is indicated for both diagnostic and therapeutic purposes.

TUBERCULOSIS OF THE LIVER.

Primary tuberculosis of the liver is hardly likely to occur. On the other hand, the liver often becomes involved in a general tubercular infection. In pulmonary tuberculosis the liver may be affected by congestion and by fatty degeneration of the parenchyma. Tubercular processes in the liver usually take the form of miliary areas in the interlobular connective tissue. In the further course they lead to interstitial hepatitis and cellular infiltration. The bacilli migrate into the liver by way of the arteries, but also through the portal vein, especially in intestinal tuberculosis. Large solitary tubercles, or accumulations of the same, are relatively rare. When superficial tubercles do occur, they may lead to perihepatitis and adhesions. It has already been stated that cirrhosis may occur as a result of hepatic tuberculosis; but clinically it occupies a less prominent position than the other features of the pathologic picture. The treatment is directed to the underlying affection.

NEOPLASMS OF THE LIVER.

Malignant Formations: Carcinoma.—Primary carcinoma of the liver is rather rare. It is estimated that 5 per cent. of all cases of hepatic carcinoma have originated in this organ. Etiologically, no further details can be adduced. It is possible that single or repeated trauma of the hepatic region favors the occurrence of primary hepatic carcinoma.

The starting point of primary carcinoma of the liver is usually the epithelium of the gall ducts, whence a cylindric epithelial form of the neoplasm develops. Scirrhus and medullary carcinomata of the liver occur as nodules of varying sizes or as infiltrations. The nodular form occurs either as a solitary roundish tumor up to the size of an infant's head, and surrounded by liver

tissue, or as numerous smaller nodules which traverse the hepatic tissue more or less densely. Adhesions with other organs occur but rarely. The infiltrating carcinomata invade diffusely the entire liver, causing considerable enlargement and imparting to the cross-section a lobular appearance.

Secondary carcinoma exhibits a great variety of form and histologic structure, according to the character of the mother tumor. The tumor cells reach the liver through the portal vein in carcinoma of the abdominal organs and particularly of the female genitals. Metastases spread by the hepatic artery from carcinoma of the esophagus, mamma, and other organs. In many cases carcinoma of the stomach, intestine and gall bladder spreads immediately to the liver. In hepatic carcinoma, aside from the growth itself, there is usually a considerable increase of connective tissue in the liver and more or less pronounced atrophy and fatty degeneration of its parenchyma.

Sarcoma. Sarcoma of the liver occurs primarily or secondarily, although the primary form is of even less frequent occurrence than primary carcinoma. The sarcomata are usually spindle-cell or round-cell forms. Melanosarcoma of the liver is a very rare occurrence. As in the case of secondary carcinoma, secondary sarcoma exhibits widely differing structures according to that of the mother growth. Like carcinoma, too, it assumes either a nodular or a diffusely infiltrated form. As a rule it grows more rapidly than carcinoma, and occurs in the young as well as in persons of more mature years.

The neoplasms develop gradually and in the early stages do not give rise to any symptoms. Later on, dyspeptic manifestations appear—anorexia, irregular stools, lassitude and weakness, nervousness and sleeplessness. In the further course, emaciation progresses rapidly. The complexion is sallow, the scleræ often show icteric discoloration, and jaundice is not a rare complication. Occasionally there is pain of varying intensity. All these manifestations may appear quite suddenly. The liver feels more or less enlarged, indurated, or nodular, according to the development and location of the tumor. The appearance of more or less pronounced jaundice depends upon the tumors spreading to the biliary ducts. When the portal circulation is impeded, ascites will develop. The spleen is usually not enlarged. Fever may result from disintegration or ulceration of the nodules. Should metastases of the peritoneum or pleura occur, they may lead to right-sided pleuritis with corresponding symptoms. Perforations may occur into the free abdominal cavity. In case of simultaneous severe cirrhosis of the liver there may be hemorrhages from the esophagus, stomach, and intestine.

The duration of the affection is estimated at from twelve to eighteen months, sarcoma usually causing a more rapidly fatal

termination than carcinoma. At the onset of the disease the diagnosis is not always easy, because, aside from other hepatic affections, hypertrophic cirrhosis, echinococci and syphilis have to be considered.

Treatment of Malignant Growths.—The treatment is purely symptomatic in the endeavor to maintain the strength of the patient as long as possible by abundant diet, to regulate the gastro-intestinal function, to relieve the pain by narcotics and cold and warm compresses, and, in the presence of jaundice, to relieve the intolerable itching of the skin. The diet should be governed according to the protective principles which apply to gastro-intestinal affections in general. Surgical treatment has been attempted, though of course with negative results. In one instance the left hepatic lobe was totally resected for carcinoma.

Benign Neoplasms.—Fibroma.—Fibroma of the liver is rare, and the tumors seldom attain to a size which is likely to cause subjective symptoms.

Angioma.—Angioma, as a rule, does not attain to more than hazel-nut size, and can only be discovered at autopsy.

Cysts.—Cysts occur here and there from congenital occlusion of the bile ducts, but are not of any clinical importance.

PARASITES OF THE LIVER.

Echinococci.—Echinococci in the human being preponderate in the liver and develop from the embryos of the *Tania echinococcus* which exists in sheep and dogs. When they find their way into the gastro-intestinal canal their outer walls are dissolved by the digestive juice, and the liberated embryo travels through the digestive organs into the circulation, whence it most frequently migrates to the liver and here grows into the fully developed parasite. Probably the chitin hooks of the embryo penetrate the tissues, and the embryos, once in the liver, develop into cysts of varying sizes. The cystic wall consists of a large number of parallel chitin layers; it is very thin, bluish white and transparent. The inner surface harbors small, bud-like structures, or scolices, from which new, sexually mature taniæ develop. The cyst is filled with a limpid, non-albuminous, uncolored liquid, which contains succinic acid. More or less numerous daughter cysts float about in this fluid, which again possess scolices with four suckers each and two coroniform rows of hooks. The size of the echinococcus cyst may vary from that of a pea to that of a man's head. The right hepatic lobe is oftener involved than the left. The shape of the liver may be considerably changed by the presence of large cysts; its increase in size may be considerable, reaching downward far into the abdomen and upward into the pleura. In some cases the echinococcus cysts look as if they were attached to the liver by

pedicles. The hepatic parenchyma in the neighborhood of the cyst atrophies more or less, according to the size of the cyst. The bile ducts are often compressed by the tumor, with resulting jaundice. The bloodvessels, too, may be so greatly compressed as to occasion portal stasis. Circumscribed or diffuse inflammation with adhesions may develop in the neighborhood of the cysts. The sac often communicates with the bile ducts, causing inflammation of the biliary system and abscesses. Occasionally a cyst may be evacuated through the bile ducts. Again, large bloodvessels may become eroded and bleed into the cyst, and phlebitis and pyemia follow. Should the echinococcus continue to grow, the hepatic parenchyma will be more and more destroyed and the neighboring organs endangered by the possibility of the cyst growing into the pleura and thus displacing the lungs and heart. Perforation may also occur into the right lung, the pericardium, the abdominal cavity, the digestive organs, the vena cava, or the renal pelvis. Perforation into the thoracic cavity is the most frequent. As the echinococcus grows older, the cystic walls continue to become thicker and firmer. If the walls are capable of great resistance, or if external obstacles prevent further growth of the echinococcus, the latter may perish. In that case the cystic contents usually become ulcerative, or form a pulpy mass consisting of fat drops, remnants of daughter cysts, detritus, and cholesterol crystals, in which the circular rows of hooks can still be found.

Symptoms.—Small cysts do not at first cause any symptoms. As they grow larger they give rise to clinical symptoms corresponding to the location of the cysts, including of course the possibility of displacement of neighboring organs. Sometimes the cyst can be directly felt. There is fluctuation and the so-called hydatid sensation, elicited on palpation when there are many daughter cysts. Occasionally there is jaundice. Patients have the sensation of fulness and tension in the hepatic region, with slight pains or none. On the other hand, when there are inflammatory processes in the cyst, considerable pain and fever are likely to arise. With the continued growth of the cyst the patient loses flesh and strength, and death ensues more or less rapidly. Spontaneous cures have been reported, and they are feasible when the cyst is evacuated into the intestine through the bile ducts. The affection usually lasts three years.

Diagnosis.—The diagnosis must exclude hepatic abscess, carcinoma of the liver, subphrenic abscess, hydronephrosis, and pleuritis. The differential diagnosis is by no means easy under certain circumstances. It is comparatively easy when by test punctures the characteristic fluid containing succinic acid and hooks can be obtained. This procedure is not devoid of danger, for the puncture may not readily close, and cases have been observed where

cystic fluid has oozed out, leading to fatal peritonitis. The presence of echinococci may be demonstrated by the complement-fixation test.

Treatment. As a prophylactic measure, infection with tænia embryos from sheep and dogs should be avoided to the utmost. The kissing of dogs, in view of the possibility of echinococcus infection, is a very dangerous habit. Internal remedies for the developed cysts, there are none. The only appropriate treatment is surgical.

Echinococcus Multilocularis.—A much rarer form than the above is an echinococcus tumor which is distinguished by the fact that the daughter cysts develop outside of the original cyst by mutual contact, assuming a honeycomb position and forming a tumor of varying size which is covered by a hard capsule and firmly adherent to the adjacent parts. The various component cysts are filled with a bile-like, yellow fluid, which seldom contains hooks and scolices. The substance becomes hard and mortar-like by inspissation. Necrotic points develop in the interior of the cyst, which is more often found in the right than in the left lobe of the liver. In this form of echinococcus cyst, jaundice is of frequent occurrence and appears early, and there is considerable connective-tissue proliferation in the hepatic parenchyma. The surface of the liver is gibbous and the organ itself considerably enlarged, with the result that the affection is very often mistaken for malignant hepatic tumor. The tumor may attain to considerable dimensions, with manifestations similar to those which occur in cirrhosis—hemorrhage from the stomach, intestine, skin and mucosa, as well as enlarged spleen. The patient's life may be spared for several years, but sooner or later the cyst proves fatal.

Other Parasites. In tropical countries, infections of the liver occur by means of the following parasites: ascarides, *Distoma hepaticum*, *Distoma hematobium* (*Bilharzia sanguinis*), *Pentastomum denticulatum*, coccidia.

FATTY LIVER.

The fatty contents of the liver, which normally amount to from 1 to 5 per cent., may pathologically increase to as much as 30 to 40 per cent. Either the fat penetrates into the liver from without (fatty infiltration) or there is fatty degeneration of the hepatic cells themselves; as a rule both modes occur simultaneously.

Etiology.—Fatty liver is not an idiopathic affection, but occurs in conjunction with numerous other pathologic conditions. In cases of general adiposity there is always fatty liver. A large number of chemical agents, such as arsenic, camphor, alcohol, carbon dioxid, mercury, and particularly phosphorus, tend to the deposition of fat in the liver, in the presence of toxemia. Fatty

degeneration in acute yellow atrophy of the liver is a well-known pathologic condition. Fatty liver also occurs in infectious diseases and chronic digestive disturbances. It is likewise a well-known fact that carcinoma, tuberculosis and leukemia are conducive to fatty liver.

Pathology.—A fatty liver retains its original shape, being uniformly enlarged in all diameters, with a smooth surface, and is yellowish-white in color. Its consistency is soft and doughy. Cross-section admits of the removal of a considerable quantity of fat with a knife. Microscopically, the periphery of the hepatic lobe and the hepatic cells themselves are seen to contain abundant quantities of fat or fat drops. The fatty infiltrated cells retain their shape, protoplasm and nucleus practically unchanged, while nucleus and protoplasm of the degenerated fatty cells are considerably damaged and inclined to atrophy.

Treatment.—The treatment is concerned with removing or modifying the underlying causes.

HEPATOPTOSIS.

Floating or migratory liver is usually a part manifestation of a general gastroenteroptosis (see page 568). Just as the stomach and intestine may prolapse by the loosening of their ligaments, so may the liver be affected by stretching and loosening of its supporting ligaments (coronary ligament). Tight lacing, pregnancy, umbilical hernia or sudden emaciation may cause relaxation of this ligament.

The movable liver can be easily demonstrated (provided the abdominal walls are not unduly thick) in the shape of a large movable and palpable tumor in the upper right abdominal region which, in the nature of things, can only be the liver. It is a particularly characteristic sign that the displaced liver sinks back into the recesses of the diaphragm when the patient assumes the dorsal decubitus. In running or walking the patient experiences a sensation of heaviness, traction and tension in the hepatic region. Other symptoms are dyspnea, constipation, meteorism, and hemorrhoids.

Treatment.—For therapeutic purposes the wearing of a well-fitting abdominal bandage is to be taken into consideration. The treatment is directed against the constitutional habitus enteroptoticus (see Chapter XXX).

NEURALGIA OF THE LIVER.

It appears that purely nervous pains may occur in the liver. The hepatic nerves emanate from the abdominal sympathetic, accompanying the hepatic arteries (hepatic plexus). As a matter

of course the diagnosis of neuralgic pains in the liver can never be made with certainty. We have reports of colicky pains in the liver, observed in hysterical individuals, which, in default of any objective findings, have been regarded as neurotic. The paroxysms of pain are quite similar to those of gallstone colic. Occasionally paroxysms of this kind occur in conjunction with menstruation as well as with various neurasthenic or neuralgic symptoms.

The object of the treatment is to remove any possible neurasthenic or hysterical underlying affection, and to apply soothing remedies, such as the bromids, morphin and heat, when paroxysm occurs.

CHAPTER XXXII.

DISEASES OF THE BILE DUCTS AND GALL BLADDER.

CHOLANGITIS; CHOLECYSTITIS; CATARRHAL JAUNDICE; HEMORRHAGE;
NEOPLASMS; DILATATION; PARASITES; GALLSTONES.

INFLAMMATION OF THE BILE DUCTS AND GALL BLADDER

Cholangitis and Cholecystitis.—Inflammation of the bile ducts and gall bladder is usually due to microorganisms—*Bacillus coli*, typhoid and paratyphoid bacilli, pneumococci, streptococci, staphylococci, cholera organisms, and tubercle bacilli. As a rule normal bile is sterile. The manner in which the choledochus inosculates into the intestine prevents the intestinal contents and intestinal bacteria from entering the duct. If the organisms mentioned above should find access to the bile ducts under pathologic conditions, either from the intestine or from the blood, certain predisposing factors must be present in order that they may display their infectious properties. Such factors are: abnormality of the epithelium of the ducts, changes in the circulation, kinking, occlusion or compression of the ducts. Inflammation of these ducts may also occur independently of bacteria, from portal stasis, passive hyperemia of the liver, or parasites invading the latter from the intestine.

Pathology.—The pathologic changes in simple cholangitis consist in swelling, hyperemia and reddening of the mucous membrane, and loosening of the epithelium. This causes more or less constriction of the ducts and, consequently, biliary stasis. As the affection proceeds the bile ducts become wholly or in part filled with a viscid or grayish-yellow mucous secretion, containing numerous desquamated cylindric epithelia. These changes are most pronounced in the larger bile ducts, and especially in the choledochus and cystic duct, and in most cases edema, hyperemia and abundant mucus are to be found at the junction of the common bile duct and the duodenum. In a case of catarrhal jaundice recently reported there was infiltration and swelling of the lymphadenoid tissue in the wall of the common bile duct similar in appearance to that seen in inflammation of the vermiform appendix. If the choledochus remains occluded for any considerable length of time this condition will lead to a more or less extensive enlargement of the tributary bile ducts and of the gall bladder. At the same time the liver is swollen. An acute catarrh will persist for

two or three weeks and undergo resolution without leaving any permanent changes. Should the affection become chronic, as in the presence of stones, parasites, neoplasms, etc., the ducts gradually become more distended and thickened, and filled with a purulent, mucous, grayish-yellow fluid. There may also be grave anatomic changes, ulcerations, etc., or even obliteration of the bile ducts. The pathologic changes in simple cholecystitis are much the same, except that occlusion of the gall bladder is more apt to occur than occlusion of the bile ducts, which tend to stretch under the inner pressure. If cholecystitis runs a chronic course it may assume a purulent, ulcerative character. There also is a possibility of the cystic contents being absorbed, leading to shrinkage and final obliteration of the gall bladder and cystic duct. Or the inflammation may spread from the mucous membrane to the other strata of the gall bladder and organs in its vicinity (stomach, intestine), causing inflammatory processes and adhesions. The pylorus is often well fixed to the right of the median line. This is the so-called "gall-bladder position of the stomach" which is easily demonstrable by the bismuth meal and the Roentgen ray. (See page 150 and Plate XXI, Fig. 2.)

Cholecystitis will often alter the outline and the size of the liver. The right lobe, as a result of the gall-bladder infection, becomes hypertrophied, either in spherical form over the site of infection or from this point downward and to the right, forming a tongue-like enlargement (known as Riedel's lobe) which may extend from the median line to the extreme margin of the liver to the right. While in pronounced cases the right lobe is distinctly palpable, that portion of the liver which lies to the left of the median line cannot be detected by the sense of touch. Sensitiveness of the affected region is manifested by rigidity of the right rectus muscle.

Inflammatory processes of the gall bladder or its adjacent structures may lead to the formation of adhesions. The most common of these adhesions implicate the duodenum or the pyloric end of the stomach, or both. It has been found that deep continuous pressure to the right of the spinal column between the seventh and eleventh ribs will induce pain in case of gall-bladder adhesion. According to Friedman, these pressure points are pathognomonic of pericholecystic adhesions with or without cholecystitis. (For Naunyn's and Murphy's signs for cholecystitis see page 619.)

Symptoms.—The symptoms of cholangitis and those of cholecystitis are very much alike. In most cases of cholecystitis the bile ducts are simultaneously affected. If the acute inflammation is at all extensive, it will cause pain in the region of the gall bladder and enlargement of this organ and of the liver. According to the intensity of the inflammation and the extent of the biliary stasis, the pains and hepatic hypertrophy will vary. At times there may be colicky paroxysms simulating gallstone colic. At any rate the

diagnosis may be doubtful until the appearance of jaundice, a characteristic symptom, which points distinctly to the bile ducts as the seat of the trouble. The pathologic picture then presented is usually characterized as catarrhal jaundice, and it is assumed that this affection is caused by the spreading of gastric or intestinal catarrh to the bile ducts. It often begins with digestive disturbances, anorexia, nausea, vomiting, eructation, irregular stools, and slight fever. Jaundice, which develops slowly, is distinctly seen in the conjunctivæ, and later also in the skin, in changing intensity. Concomitant manifestations, consisting of retarded pulse and itching of the skin, will make their appearance. In many cases, however, gastric and intestinal manifestations are entirely absent, and in these cases the etiology of the jaundice is not always clear. There is a clear connection between cholecystitis and myocardial incompetence. A weak myocardium is the rule in all gall-bladder affections. The assumption has recently been favored that there is such a thing as functional jaundice—that the pigment components of the bile and the components which cause pruritus are not produced at the same time or place, perhaps running in different directions, with the consequence that the entire bile-producing apparatus and the hepatic function are interfered with. This conclusion has been drawn from the fact that at times itching precedes jaundice by several days or persists after the jaundice has subsided, and from the further fact that in some cases of jaundice there is no itching, the substances that cause it having, presumably, not been produced at all. It is also a striking fact that catarrhal jaundice not infrequently occurs endemically. Taking into consideration the lymphadenoid affection of the choledochus referred to above, this might give color to the idea that jaundice originates hematogenously from infection, like the frequent occurrence of appendicitis after tonsillitis. As the catarrh subsides, the symptoms likewise gradually disappear, jaundice and its accompaniments recede, the feces (which were clay-colored) resume their normal color, and the enlarged liver undergoes involution. However, nearly every prolonged case of jaundice causes weakness, emaciation, and anemia—a fact which is explained by the injurious influence exerted by the biliary acids upon the red blood-corpuscles, depriving them of hemoglobin.

When there is biliary-duct obstruction inducing the back flow of bile into the liver, serious damage is done to the liver cells. Necrosis and connective tissue change take place. It is assumed that the destruction of the liver tissue depends upon the chronicity of the biliary stasis. The longer a clinical jaundice continues, the more damage is done to the liver.

Among the sequelæ of catarrhal jaundice, gallstone formation ranks first, and there is no doubt that concretions are often formed in the bile in cholangitis. A serious complication consists in the

two or three weeks and undergo resolution without leaving any permanent changes. Should the affection become chronic, as in the presence of stones, parasites, neoplasms, etc., the ducts gradually become more distended and thickened, and filled with a purulent, mucous, grayish-yellow fluid. There may also be grave anatomic changes, ulcerations, etc., or even obliteration of the bile ducts. The pathologic changes in simple cholecystitis are much the same, except that occlusion of the gall bladder is more apt to occur than occlusion of the bile ducts, which tend to stretch under the inner pressure. If cholecystitis runs a chronic course it may assume a purulent, ulcerative character. There also is a possibility of the cystic contents being absorbed, leading to shrinkage and final obliteration of the gall bladder and cystic duct. Or the inflammation may spread from the mucous membrane to the other strata of the gall bladder and organs in its vicinity (stomach, intestine), causing inflammatory processes and adhesions. The pylorus is often well fixed to the right of the median line. This is the so-called "gall-bladder position of the stomach" which is easily demonstrable by the bismuth meal and the Roentgen ray. (See page 150 and Plate XXI, Fig. 2.)

Cholecystitis will often alter the outline and the size of the liver. The right lobe, as a result of the gall-bladder infection, becomes hypertrophied, either in spherical form over the site of infection or from this point downward and to the right, forming a tongue-like enlargement (known as Riedel's lobe) which may extend from the median line to the extreme margin of the liver to the right. While in pronounced cases the right lobe is distinctly palpable, that portion of the liver which lies to the left of the median line cannot be detected by the sense of touch. Sensitiveness of the affected region is manifested by rigidity of the right rectus muscle.

Inflammatory processes of the gall bladder or its adjacent structures may lead to the formation of adhesions. The most common of these adhesions implicate the duodenum or the pyloric end of the stomach, or both. It has been found that deep continuous pressure to the right of the spinal column between the seventh and eleventh ribs will induce pain in case of gall-bladder adhesion. According to Friedman, these pressure points are pathognomonic of pericholecystic adhesions with or without cholecystitis. (For Naunyn's and Murphy's signs for cholecystitis see page 619.)

Symptoms.—The symptoms of cholangitis and those of cholecystitis are very much alike. In most cases of cholecystitis the bile ducts are simultaneously affected. If the acute inflammation is at all extensive, it will cause pain in the region of the gall bladder and enlargement of this organ and of the liver. According to the intensity of the inflammation and the extent of the biliary stasis, the pains and hepatic hypertrophy will vary. At times there may be colicky paroxysms simulating gallstone colic. At any rate the

diagnosis may be doubtful until the appearance of jaundice, a characteristic symptom, which points distinctly to the bile ducts as the seat of the trouble. The pathologic picture then presented is usually characterized as catarrhal jaundice, and it is assumed that this affection is caused by the spreading of gastric or intestinal catarrh to the bile ducts. It often begins with digestive disturbances, anorexia, nausea, vomiting, eructation, irregular stools, and slight fever. Jaundice, which develops slowly, is distinctly seen in the conjunctivæ, and later also in the skin, in changing intensity. Concomitant manifestations, consisting of retarded pulse and itching of the skin, will make their appearance. In many cases, however, gastric and intestinal manifestations are entirely absent, and in these cases the etiology of the jaundice is not always clear. There is a clear connection between cholecystitis and myocardial incompetence. A weak myocardium is the rule in all gall-bladder affections. The assumption has recently been favored that there is such a thing as functional jaundice—that the pigment components of the bile and the components which cause pruritus are not produced at the same time or place, perhaps running in different directions, with the consequence that the entire bile-producing apparatus and the hepatic function are interfered with. This conclusion has been drawn from the fact that at times itching precedes jaundice by several days or persists after the jaundice has subsided, and from the further fact that in some cases of jaundice there is no itching, the substances that cause it having, presumably, not been produced at all. It is also a striking fact that catarrhal jaundice not infrequently occurs endemically. Taking into consideration the lymphadenoid affection of the choledochus referred to above, this might give color to the idea that jaundice originates hematogenously from infection, like the frequent occurrence of appendicitis after tonsillitis. As the catarrh subsides, the symptoms likewise gradually disappear, jaundice and its accompaniments recede, the feces (which were clay-colored) resume their normal color, and the enlarged liver undergoes involution. However, nearly every prolonged case of jaundice causes weakness, emaciation, and anemia—a fact which is explained by the injurious influence exerted by the biliary acids upon the red blood-corpuscles, depriving them of hemoglobin.

When there is biliary-duct obstruction inducing the back flow of bile into the liver, serious damage is done to the liver cells. Necrosis and connective tissue change take place. It is assumed that the destruction of the liver tissue depends upon the chronicity of the biliary stasis. The longer a clinical jaundice continues, the more damage is done to the liver.

Among the sequelæ of catarrhal jaundice, gallstone formation ranks first, and there is no doubt that concretions are often formed in the bile in cholangitis. A serious complication consists in the

agglutination of the common bile duct, which may occur through loss of its epithelium or as a result of ulcerative processes, leading to chronic biliary stasis. This may cause an enormous dilatation of the bile duct and gall bladder, serious injury to the liver, emaciation, and cachexia. If the obliteration is confined to the cystic duct, there will be dropsy of the gall bladder; if branches of the hepatic duct are involved, there may be partial atrophy of the hepatic tissue. The test-diet stool is characteristic (see Chapter IV). When there is an obstruction of the common duct the duodenal contents are typical (see page 109).

The affection lasts from three to four weeks, in light cases from ten to fourteen days; cases, however, have been observed which continued for three or four months. As a general rule the prognosis is good, unless the chronic changes above referred to should supervene.

Cholemia is occasionally a family affection, but the pathology of this form is not clearly understood. The usual cause is an angiocholitis, or some obstacle preventing the normal flow of bile, which induces hypertrophy of the liver, with urobilinuria. Familial cholemia can be separated into two stages: the first being a long one of compensation, well borne by the patient, the second stage one of broken compensation and resulting complications. During the first stage hemorrhages often occur, as epistaxis and menorrhagia. The functional disturbance of the liver is apt to cause the violent attacks of migraine which are seen in cholemia.

Diagnosis.—Examination of the duodenal contents should be made in all cases of suspected cholangitis and cholecystitis (see pages 104 and 109). It furnishes direct diagnostic evidence of the beginning of biliary stasis, the precursor of biliary disease. To drain the biliary system, the duodenum should be douched with 90 C.c. of a 30-per-cent. solution of magnesium sulphate, which relaxes the sphincter of the common duct and contracts the gall bladder. In a few minutes there is a free flow of bile in the duodenum. Lyon¹ demonstrated that the first bile collected is from the common duct, cystic duct, and hepatic ducts. It soon becomes vividly golden-yellow in color and of a syrupy consistence. This is followed by a second and darker bile, presumably from the gall bladder; and then comes a thinner common yellow bile that has been freshly secreted by the liver cells. Any deviation from this normal secretion means pathological change in the biliary system. It is therefore important that attention be given to the gross appearance of the bile, such as color, consistency, viscosity, transparency, and turbidity. Especial study should be made microscopically for the presence of bile-stains, epithelium, pus, leukocytes, red blood-corpuscles, crystals, concretions, mucus, and bacteria.

¹ B. B. Vincent Lyon: The Need of Early Diagnosis and Treatment of Cholangitis, Cholecystitis and Cholelithiasis, *Annals of Medicine*, July, 1920.

In a report of his pathologic findings in 1000 gall-bladder cases Smithies declares that in nearly 50 per cent. pericholecystic adhesions were found, in which the duodenum was usually involved, inducing displacement and distortion of the latter. The Roentgen ray will not always show this abnormality, because the opaque meal in the stomach overlaps the descending and transverse portions of the duodenum. Palefski¹ utilizes an especially long duodenal tube under such conditions to visualize the duodenum by means of the Roentgen ray. Displacements and angulations due to periduodenal adhesions can thus be recognized. The course of the duodenal tube follows the lesser curvature of the stomach through the pylorus into the jejunum. Normally the roentgenogram shows the tube in the duodenum as an unaltered horseshoe-shaped curve. Distortion of this duodenal curve indicates displacement and adhesions of the duodenum suggestive of gall-bladder pathology.

Traction on the umbilicus in the direction of the pubes often induces pain over the gall-bladder region (see page 619.)

Hemocones.—Hemocones provide an indirect means of ascertaining the existence of bile acid retention. They are for the most part granules of fat, emulsified in the small intestine, absorbed and carried into the general circulation through the thoracic duct. They are easily demonstrable by the ultramicroscope as tiny sparkling granulations, animated by constant well-marked Brownian movement. To detect them, the patient is given an ounce of butter on a slice of bread. Three hours later a drop of blood is taken from the finger, placed on a slide and gently squeezed with a cover-glass. The hemocones can be readily seen in the open spaces with the ultramicroscope. They are found in the blood for four or five hours in proportion to the amount of fat ingested. In cases of obstruction, the bile not entering the duodenum, no hemocones are found in the blood after the ingestion of butter. When it is desired to ascertain the state of the secretion of bile salts, the finding of hemocones indicates that the ingested butter has been absorbed after due elaboration by the bile salts. A negative result would point to defective absorption of fats and inferentially to the lack of bile salts in the duodenum.

Treatment. Experience has shown that catarrhal jaundice is most favorably influenced by Carlsbad mineral waters, though there is no evidence that these waters have any cholagogue action; their beneficial effect is probably due to their anticatarrhal properties. Furthermore, the alkalis contained in the Carlsbad waters will protect the red blood-corpuscles from the hemolytic action of the biliary acids in jaundice by raising their power of resistance. The Carlsbad cure is best taken in Carlsbad itself, but may also be carried out at home with Carlsbad Muhlbrennen or Schloss-

¹ I. O. Palefski. Intubation and Visualization of the Duodenum with the Duodenal Tube, *Journal of the American Medical Association*, December 4, 1920

brunnen. The treatment should not commence until after gastric or intestinal symptoms have been relieved. It is as follows: In the morning 200 Cc. (7 ounces) of the water, heated to 90° F., is taken upon an empty stomach, followed by the same quantity twenty minutes later. Four hours after the midday meal the same quantity is ingested at the same temperature, and immediately before retiring 150 Cc. (5 ounces) at a somewhat lower temperature. After a week the temperature of the water may be raised to 95° to 100° F. If indicated, and especially in constipation, 4 Gm. (3j) of Carlsbad sprudel salt or Carlsbad effervescent salt is added to the first glass of water. Sodium phosphate, sodium sulphate or magnesium sulphate can be given with the same benefit (see page 284). Small doses of pulvis rhei compositus likewise have a favorable effect. While taking this treatment the patient should not walk much. The treatment should be kept up for two or three weeks after the jaundice and the swelling of the liver have disappeared, and repeated about six months later. In regard to jaundice, testing of the urine for urobilinogen is of practical importance, because this substance is often demonstrable long after urobilin has ceased to be. The Carlsbad cure presupposes normal gastric motility, and any changes in the circulation demand caution. Both quantity and temperature of the water should be kept within moderate limits, especially when the blood-pressure is high.

Einhorn found that glycerin, given in teaspoonful doses three times daily, exerts an antiputrefactive action on the bile. This was proved by a study of the bile after its removal through the duodenal tube. Patients to whom glycerin has been given furnish a bile that can be kept from one to two days. Without this medication the duodenal secretion after exposure to the air for a few hours begins to decompose and in the course of six hours develops a putrid odor.

The following may be given with advantage:

	Gm. or Cc.	
R—Sodu bicarbonatis	8 0	3ij
Glycerum	60 0	3ij
Aquæ destillatæ	150 0	3v

Misce

Sig. Tablespoonful three times a day, half an hour before meals.

Salicylic acid and hexamethylenamin have a distinct disinfecting action upon the bile and probably a direct cholagogue effect. The polyvalent combined bacterial vaccines are valuable adjuvants.

The local application of a 25-per-cent. solution of magnesium sulphate to the intestinal mucosa causes a relaxation of the muscular wall. On the introduction of one ounce of this solution through the duodenal tube into the duodenum, the sphincter of the common bile duct relaxes, allowing free flow of bile. Duodenal lavage with a solution of magnesium sulphate should be practiced in all these cases. Thus non-surgical drainage of the gall bladder

and bile ducts gives most gratifying results. This is a simple process, since we are able to use the duodenal tube to advantage. With its aid we can apply medication directly to the mucous membrane of the duodenum (see page 104). By duodenal lavage the biliary apparatus can be thoroughly drained (see page 105).

The diet should be absolutely free from irritating constituents. Carbohydrates should be given in liberal quantities (rice, farinaceous food, farinaceous infant foods, dextrinized flour). As to fats, none but emulsified or easily emulsifiable fats are allowed—which means small quantities of butter and milk, because other neutral fats cannot be emulsified in the absence of the biliary flow. Nor is it advisable to allow large quantities of meat, because putrefactive processes may easily develop in the intestine in stasis of the septic bile and accompanying disturbance of pancreatic function. For this reason none but tender meat (veal, chicken, pigeon, sweetbread) may be given. Pancreatic preparations may be indicated as adjuvant treatment (see page 262). Vegetables are allowed in purée form and finely divided. The meals should be small, but frequent.

Patients should have much rest in bed, especially in the first stages of the affection and according to the degree of its intensity. Hot cataplasms are applied to the hepatic region. The unpleasant itching is at times relieved by ablutions with diluted vinegar, lemon juice, chloroform, spirit of menthol, or warm baths. Occasionally the itching ceases after a hypodermic injection of pilocarpin.

As a palliative measure, Anderson's powder is useful:

	Gm. or Cc.	
R—Camphoris	6 0	3iss
Zinci oxidi	15 0	3i+ss
Amyli	30 0	3j
Miscc.		

Sig.—Dust lightly over skin with a powder puff.

Suppurative Cholangitis and Cholecystitis.—**Etiology.**—This originates from the same causes as simple catarrhal inflammation and occurs when the latter takes a grave course. It may, however, also occur in the course of serious infectious diseases (cholera, sepsis, typhoid), or it may spread from neighboring inflammations and ulcerations to the bile ducts or gall bladder. Mechanical causes such as gallstones, parasites or wounds are often the predisposing factor, enabling the microorganisms to gain a foothold.

Pathology.—The gall bladder is affected most frequently. It, as well as the diseased bile duct, is dilated and filled with a mucopurulent or ichorous fluid. The mucous membrane is hyperemic, incrassated and hemorrhagic, while fibrinous inflammations with necrosis of the mucosa and ulceration may supervene. The ulcerations may permeate the entire wall of the gall bladder or ducts, leading to abscesses of the liver, adhesions to the neighboring

parts, and perforation of the gall bladder. In the course of destructive processes in the wall of the gall bladder, biliary effusion into the peritoneal cavity is not infrequent, causing biliary peritonitis. At autopsy, microscopic examination is often necessary for the detection of the minute defects in the wall of the gall bladder or of the larger ducts through which the bile has percolated. When the gall bladder is filled with pus and dilated, we have to deal with an *empyema* of the gall bladder. The finding of pus after aspirating the duodenal contents with the duodenal tube assists in an early diagnosis, providing the gall bladder is able to discharge a specimen of its contents into the duodenum.

Symptoms.—At the onset there is the same picture as in catarrhal inflammation. The gravity of the affection does not become apparent until chills and fever occur. Then there is enlargement of the gall bladder and pain in the hepatic region, although these symptoms are also present in other affections of the gall bladder, calculi, parasites, and tumors. For this reason the diagnosis is always uncertain until abscesses, peritonitis, perforation or pylephlebitis complicate the picture. Manifestations of this kind, of course, render the prognosis grave.

Treatment.—The treatment is directed largely to checking the inflammatory process as far as possible by rest in bed, application of ice, mild lactovegetable diet, and light laxatives. When the fever is pronounced, antipyretic remedies are prescribed. In collapse, alcohol and the injection of camphor are to be considered. In all cases, however, where there is a suspicion of infectious cholecystitis, surgical measures should be resorted to as soon as possible, to prevent necrosis and perforation of the wall of the gall bladder.

HEMORRHAGE INTO THE BILE DUCTS

Hemorrhages into the bile ducts occur as a result of chronic hyperemia, necrosis of the mucosa, or trauma. Aneurysms of the hepatic artery have also been observed to perforate into the bile ducts. Any treatment in such cases is out of the question, because they cannot be diagnosticated during life.

NEOPLASMS OF THE BILE DUCTS AND GALL BLADDER.

The most common of these are carcinomata, while in rare cases we find fibroma, sarcoma, cysts, papilloma, and tubercles. Carcinoma occurs in the bile ducts in the form of circumscribed nodules of the mucosa or as a diffuse infiltration of the latter. The mucous membrane may be intact, but frequently it is ulcerated. As the neoplasm continues to proliferate, the walls of the bile ducts become rigid and thickened, and the lumen constricted or occluded. Gallstones are present in many cases. Carcinoma of the gall bladder occurs in a similar way and is nearly always associated with stone

formation, the latter probably being antecedent to the development of the neoplasm. Carcinoma of the choledochus and of the papilla of Vater has likewise been observed.

Symptoms.—Neoplasms of the bile ducts do not cause any manifestations unless they lead to occlusion of the larger ducts and to jaundice. This is followed by rapidly increasing manifestations of biliary stasis, swelling of the liver, and cachexia. Carcinoma of the gall bladder is usually associated with pain in that region, which is often persistently present and in many cases resembles gallstone colic. Gradually, a hard growing tumor can be palpated in the gall bladder. In carcinoma of the choledochus, jaundice occurs without any paroxysms of pain. Women are more frequently the subjects of these malignant growths than men.

Diagnosis.—Carcinoma of the bile ducts cannot be diagnosticated with certainty, but its presence may be assumed when chronic jaundice is associated with carcinomatous cachexia. The affection may last for years. In carcinoma of the gall bladder the presence of the distinct growth suggests the diagnosis, although there is a possibility of the growth having started from other organs in the vicinity of the gall bladder without giving any positive evidence of the fact. A normal gall bladder does not lose its elasticity. When the wall of a gall bladder has become infiltrated with leukocytes and thickened, it is non-distensible. Infection lessens the distensibility. So when there is complete obstruction of the bile duct due to a carcinoma at or near the duodenal papilla, or at or near the pancreas, the gall bladder is distended and a tumor is palpable. Courvoisier's law applies here, that if there is obstruction at the common duct due to malignant disease a tumor will be found in the region of the gall bladder. When the gall bladder is small and jaundice is present the obstruction is due to stone. The hemolytic, antitryptic or miostagmin reaction may aid in the diagnosis (see page 543). The Roentgen ray is often of great assistance in the diagnosis (see Chapter V).

Treatment.—This is surgical. The gall bladder degenerated by carcinoma, together with the adjacent hepatic tissue, must be removed. Surgery offers no hope of cure, however, when there are hepatic metastases or when the portal lymph gland is already affected. Removal of carcinoma of the choledochus or of the papilla of Vater has been successful in a few cases. Aside from this there is nothing left but resort to palliative operations, such as a biliary fistula outward or a fistula between the gall bladder and the small intestine.

DILATATION OF THE BILIARY ORGANS.

Dilatation of the bile ducts is always a consequence of their more or less complete occlusion. Among possible causative factors

the following are to be considered: gallstones, parasites, chronic inflammation with obliteration or cicatricial stenosis of the ducts, congenital occlusion of the choledochus, neoplasms, aneurysms of the hepatic artery, fecal stasis, and neoplasms outside the liver.

Hydrops and Empyema. In hydrops the arrested bile is gradually absorbed, being replaced by a serous transudate from the bloodvessels. A pear-shaped tumor will slowly grow and occasionally attain to gigantic size. Empyema of the gall bladder will follow when the cystic contents undergo infection in any way.

Symptoms.—Hydrops of the gall bladder gives rise to but few subjective manifestations—slight pain and pressure in the region of the gall bladder. The course is chronic, with the result that the condition remains unchanged for a long time, causing but slight inconvenience to the patient. In empyema, fever will develop, with chills and other manifestations of sepsis.

Treatment.—Physical rest, light diet and mild laxatives may arrest the progress of hydrops. Large tumors causing much discomfort, with an impending rupture, demand surgical intervention (cholecystotomy); as also does the mere suspicion of empyema.

PARASITES OF THE BILE DUCTS.

Echinococcus cysts are among the parasitic affections most frequently met with in the bile ducts; also ascarides and *Distoma hepaticum*. Ascarides and echinococci may, by causing occlusion of the bile ducts, give rise to grave consequences. Ascarides invade the bile ducts from the duodenum. The changes thereby produced correspond to those of grave suppurative inflammation. A cure may be effected if the ascarides find their way out into the intestine through the choledochus (see page 801).

Diagnosis.—The diagnosis can only be made with great reserve, it being necessary to demonstrate the presence of ascarides aside from the symptoms of suppurative cholangitis.

Treatment.—The treatment has then to be directed to the removal of the worms.

GALLSTONES.

Cholelithiasis.—Gallstones are concretions which form in the gall bladder and bile ducts. They are of most varied shape, size and composition, and may be divided into pure cholesterol stones, lamellated cholesterol stones, pure bilirubin limestones, bilirubin limestones mixed with cholesterol, and stones consisting of carbonate of lime. The number of stones in a single case varies from one to one hundred or more. They are found in the liver, in the narrowest bile passages where they sometimes appear as minute black concretions, in the hepatic duct or its branches, in the gall bladder, in the cystic duct, in the choledochus, and in the diver-

ticulum of Vater. The stones found in the cystic and common bile ducts usually originate in the gall bladder. Gallstones are also found in the intestine, which they may enter from the choledochus or by perforation of the gall bladder; they have been found in the urinary bladder and urethra after perforations. The size of the stones varies; some are no larger than the finest sand, others may be as large as a hen's egg. They are usually round or oval. When many stones lie close together, they will flatten by contact and become polyhedral (faceted). Age plays a considerable part in the development of gallstones; they occur at any age, but most frequently between the ages of fifty and sixty. The proportion of cases in the male as compared with the female sex is as 2 to 3.

As to the origin of gallstones, the prevailing modern theory is as follows: The presence of bile stasis is a prerequisite, whether from mechanical injuries (tight lacing, pregnancy) or from some precedent affection of the bile ducts or liver. The arrested bile may exert a directly injurious influence upon the epithelia of the mucosa by causing a kind of desquamative catarrh and detaching cellular detritus containing cholesterol, around which the gallstones form, or by causing the formation of an albuminous and calcareous secretion of the mucosa, under the influence of which bilirubin lime is deposited from the bile. On the other hand, bile stasis may favor the access of microorganisms from the intestine or the blood, and the development of infectious cholangitis, the affected mucosa then giving rise to the formation of gallstones in the manner just described. As a matter of fact, it is by no means rare for the anamnesis of gallstone patients to include a history of previous jaundice. It is also a well-known fact that gallstones frequently occur in the wake of acute infectious diseases, especially influenza and typhoid fever. According to another theory, the biliary salts which keep the cholesterol of normal bile in solution are decomposed by the bacteria, precipitating the cholesterol. Other authors maintain that genuine stone formation can only occur when salts and cholesterol are separated from the bile together with protein substances, protein being the foundation of all concretions developed in the organism. It is finally to be considered that in gallstone patients hereditary factors often play a rôle; in some families every member suffers from this disease. It is also a noteworthy fact that biliary and renal calculi are often observed in one and the same patient. These facts admit of the possibility that abnormal metabolic processes or changed consistency of the blood may have to be taken into account as causative factors. Cholelithiasis may result from an excess of cholesterol esters in the digestive tract or in the blood (cholesterolemia), due to defective metabolism of ingested lipoids, or to an excess of these in the diet. In obstructive jaundice the cholesterol content of the blood is markedly increased and bears a definite relationship to the intensity of the jaundice.

In conditions associated with diseases of the liver the cholesterol content of the blood is usually increased. In hemato-genous jaundice there is no increase of blood cholesterol.

Symptoms.—A great many individuals harbor gallstones without ever suffering any inconvenience therefrom. The stones remain at rest in the ducts or bladder. Others experience slight pain or pressure in the hepatic region and slight gastric and abdominal symptoms, which, however, do not lead to colics or other serious trouble. The great majority of patients, however, suffer from pronounced gallstone colic; the stones wander from the bile ducts or gall bladder through the choledochus into the duodenum, causing more or less severe pain. The migration of stones is favored by lifting heavy weights, concussion of the body, psychic irritation, or partaking of cold beverages. The paroxysms may run a light course, or lead to excruciating pain with general convulsions and loss of consciousness. They vary in duration. Occasionally a paroxysm will last for several days, with short intervals. The attack usually begins a few hours after a heavy meal, and most often in the evening or at night. The region of the gall bladder becomes very painful and the corresponding part of the abdominal wall very tense. The paroxysm will not terminate until the stone has fallen back into the gall bladder or entered the intestine. A very characteristic sign of gallstone paroxysms is almost complete insensibility to pain in the region of the gall bladder immediately after the paroxysm has ceased. If the paroxysm has been unsuccessful, having failed to remove the stone, fresh paroxysms may occur without interruption until the stone or stones have been evacuated. Inflammatory manifestations and fever are absent in uncomplicated paroxysms. Should fever occur, it would point to simultaneous inflammatory processes in the bile duct. When a paroxysm has come to an end, patients may remain unmolested for a time, perhaps for many years, or even permanently. In other cases, however, the concretions will lead to acute, subacute or chronic irritative and inflammatory conditions. These patients will suffer continually from more or less pain, pressure and tension in the hepatic region, and are often driven to bed as a consequence; the acute affection has developed into chronic cholecystitis, adhesions have been formed with the surrounding parts, or stones have become encapsulated and continually irritate the surrounding parts, causing chronic inflammation and ulceration. In the event of rupture or perforation of the gall bladder, death may ensue.

Diagnosis.—Roentgen-ray examination has given satisfactory results in 50 per cent. of the cases in which it has been employed (Plate XXI, Fig. 2). The typical gallstone paroxysm is unmistakable in its course and in the pain, which is confined to the right side. Jaundice need not be present. Gallstone paroxysms and chronic gallstone affections may be mistaken for gastric crises, lead colic,

intercostal neuralgia, appendicitis, epigastric hernia, renal or pancreatic calculi, acute or chronic pancreatitis, hepatic abscess, inflammation of the gall bladder from other causes, abdominal pain, gastric or duodenal ulcer. In these cases it is not easy to make a safe diagnosis. In many cases the anamnesis gives a correct clue. It should also be remembered that in gallstone affections there is often carcinoma of the bile ducts. The jaundice met with in malignant disease is distinguished by the absence of colic and pain, gradual onset (without remissions) deepening day by day, the skin finally becoming greenish-yellow. Courvoisier pointed out many years ago that atrophy of the gall bladder is the rule when the common duct is occluded from within, as by a gallstone. When a gall bladder is distended, associated with jaundice, the pressure is usually outside of the duct. In approximately 85 per cent. of cases of stone in the common duct the gall bladder is contracted. Hypercholesterolemia is of some diagnostic importance, especially when surgical measures are contemplated, though it must not be forgotten that the cholesterol content of the blood varies widely in other conditions, such as pregnancy, gastric or duodenal ulcer, chronic nephritis, or even simple inanition.

Naunyn's sign for cholecystitis consists of deep tenderness when at the end of a full inspiration the examiner's fingers are thrust upward beneath the costal arch at the outer limit of the right epigastrium.

Murphy's sign of gall-bladder disease consists in the inability of the patient to take a deep inspiration when the examiner's fingers are hooked up deep beneath the right costal arch below the hepatic margin.

Examination of the duodenal contents removed with the duodenal tube is often helpful in the diagnosis. Occasionally small stones are thus discovered. The finding of gall sand and a gritty feeling experienced by the examining finger are very suggestive. Microscopically large masses of crystals and bile salts are frequently revealed (see page 109).

Treatment.—Gallstone colics demand rapid help. Unless the attack is slight, the pain should be relieved by anodynes and narcotics, the best being a liberal subcutaneous injection of morphin—0.015-0.03 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain). Atropin may be combined with the morphin. Opium, pantopon and codein do not have the same prompt effect as morphin. The patient is brought to bed, and hot compresses (poultices, electric pad, hot-water bottle) are applied to the hepatic region. A prolonged hot bath may also be given. Olive oil will often mitigate the pain (see page 273). Vomiting is controlled by ice chips or chloroform; lavage of the stomach with hot water and the ingestion of large quantities of hot liquids have a favorable effect. In collapse the usual analeptics

are administered. In this way it is possible to help patients over a serious attack with comparative ease. The patient remains in bed until the pain and pressure in the hepatic region have disappeared. During the following few days it will be opportune to prescribe mild laxatives. When there are no further signs of paroxysms and no other symptoms, patients may resume their occupation.

If gallstone paroxysms have been definitely demonstrated, if the paroxysms are of frequent recurrence at regular or irregular intervals, or if there are permanent symptoms of chronic inflammatory conditions in the biliary system, the general treatment of gallstone affections has to be instituted. The object of this treatment is not the removal of the gallstones, for there are no medicinal means at our disposal to accomplish such a purpose. The principal object, rather, is to establish a stage of latency—rest in the entire system of bile channels. If this can be accomplished, the inflammatory changes in the gall bladder and bile ducts, which are present in the majority of all cases, may undergo a complete cure. The first rank in the treatment of gallstone disease is held by the Carlsbad mineral waters. These are best taken at the springs, but, other conditions being favorable, may also be taken at home. All cases of cholelithiasis are suitable for this treatment, but preferably those which have exhibited symptoms for a short time only. In these cases the Carlsbad cure may also by way of prophylaxis prevent further concretum-formation. The favorable action of the Carlsbad waters is partly explained by their anticatarrhal effect. It may further be assumed that these hot mineral waters improve the circulatory conditions in the hepatic region, thereby increasing the fluidity of the bile and inhibiting stone-formation, and that the more fluid bile will carry away small concretions. All the saline laxatives act in a similar manner and are of great value (see page 284); it is said that they cause cholesterolemia to disappear, should such exist. If the gastric motility is normal the Carlsbad treatment can be given as described on page 611. While taking the cure it is important that the patient rest reclining, to protect the body from unnecessary concussion. Before and after dinner one and one-half to two hours should be spent in the dorsal decubitus, during which time hot poultices or compresses may be applied to the hepatic region. The rest cure should be continued to some extent for several months after the mineral-water treatment has been discontinued—careless movements, long walks, etc., being avoided. The transition to a more strenuous life should be gradual.

While taking the treatment the diet should be absolutely bland. All food should be carefully comminuted, and the meals should be small and frequent. A diet rich in carbohydrates is generally to be commended. As inundation of the liver with nitrogenous sub-

stances is to be avoided, the quantity of meat should be restricted. Foods rich in fat, as eggs, butter, cream and fish, tend to increase the cholesterolemia and should be rigidly excluded in cases showing an excessive amount of cholesterol in the blood. Fresh bread, bakers' wares prepared with yeast, raw fruit, and coarse vegetables are disallowed. Alcohol is allowed in the form of a light white wine, but not if there is hyperacidity. Cold foods and beverages, especially those cooled on ice, are to be scrupulously avoided.

Medication.—Cholagogues and antiseptics: Podophyllin, 0.03 to 0.05 Gm. ($\frac{1}{2}$ to 1 grain) in pills; calomel; belladonna; salicylic acid; sodium salicylate, 0.5 to 1 Gm. ($7\frac{1}{2}$ to 15 grains); and hexamethylenamin, 0.5 Gm. ($7\frac{1}{2}$ grains) several times daily. There are many proprietary combinations, such as: probilin pills (consisting of salicylic acid, sodium oleate, menthol and phenolphthalein); eunatrol, containing sodium oleate; and agobilin, containing cholic acid, strontium and phenolphthalein. Chologen, as recommended by Glaser, consists of calomel, podophyllin, camphor, caraway and aromatics, and has given good results in some cases. It is supposed not only to increase the quantity of bile, but to increase the proportion of the normal constituent which is said to be a gallstone solvent. It is given in tablet form before each meal, the dose being increased or decreased according to the movement of the bowels. According to recent clinical experience, during the first ten days one or two tablets of No. 1 are taken before the morning and midday meals. During the next forty days one or two tablets of No. 1 are taken before the morning and midday meals and two tablets of No. 2 are taken before the evening meal. During the next ten days one tablet of No. 3 is taken before the morning, midday and evening meals. If an attack of gallstone colic is threatened, three tablets of No. 2 should be taken at once. When the inflammation in the bile ducts or gall bladder is very severe, with fever and chills, the systematic administration of calomel, 0.1 Gm. ($1\frac{1}{2}$ grains) three times a day, kept up for at least four weeks, may save the patient an operation. Generally, the course of cholelithiasis seems to be benefited by the use of mild laxatives (see Chapter XIV) from the very beginning of the treatment, since easy stools provoke secretion of bile. Partaking of frequent, easily digestible meals has a distinct cholagogue effect. In hyperacidity the administration of the alkalis is beneficial. For the relief of the colic, papaverin or benzyl benzoate can be employed (see page 276).

Non-surgical Biliary Drainage.—The biliary apparatus can be drained by the use of the duodenal tube (see Chapter III). Meltzer found that a 25-per-cent. solution of magnesium sulphate in the duodenum produced complete relaxation of the intestinal wall. It is thus possible, by the use of magnesium sulphate introduced directly into the duodenum, to relax the sphincter of the common

duct and permit the ejection of bile and even the removal of a calculus of moderate size wedged in the duct in front of the papilla of Vater.

Surgical Treatment.—Cases of gallstone colic which occur at certain intervals but pass without unduly interfering with the patient's occupation do not require an operation. Exaggerated frequency of the attacks and considerable interference with work may render an operation desirable. The prognosis of these operations (cholecystotomy, cholecystectomy) is generally good; the mortality is 3 per cent. in the absence of inflammation, 5 to 10 per cent. when there is suppurative inflammation. Cholelithiasis associated with cholemia and inflammation is an absolute indication for operation. Occlusion of the choledochus by stones demands operation, unless the concretions are evacuated at an early stage. Fever, chills and septic manifestations demand prompt surgical intervention, although a delay of a few weeks is permissible when the course is chronic. It should be remembered, however, that jaundice involves danger and that chronic cholangitis may lead to abscess of the liver. Perforation of pus and stones into the peritoneal cavity is an urgent indication for operation. We now know that various diseases are intimately related to one another. The gall bladder and appendix have been found involved at operation for ulcer of the stomach and duodenum. It is said that in 30 per cent. of all diseases of the gall bladder there is an infected appendix. Surgeons should always examine the appendix when they operate on the gall bladder.

CHAPTER XXXIII.

DISEASES OF THE PANCREAS.

PANCREATITIS; ACHYLIA; HEMORRHAGE; NECROSIS; CYSTS;
TUMORS; CALCULI.

INFLAMMATION OF THE PANCREAS.

MANY acute and chronic affections of the pancreas require surgical treatment. Internal treatment plays rather an adjuvant but by no means unimportant rôle, especially in regard to diet, as will be explained. Very few pancreatic affections are directly benefited by medicinal treatment; these include pancreatic fistulæ, functional disturbances of the pancreatic secretion secondary to achylia gastrica, cholelithiasis or cholecystitis, or primary (achylia pancreatica) and chronic pancreatitis—notably the last named. Chronic pancreatitis will be dealt with first, because its clinical manifestations, especially in regard to digestion, are more or less met with in other chronic pancreatic affections (tumors, calculi), and because the indications for internal medication can be clearly deduced from its clinical course.

Chronic Pancreatitis. This affection is by no means as rare as is often supposed, and, with our modern knowledge, can in most cases be correctly diagnosed, although the anamnesis does not furnish any typical data. It usually begins with an ill-defined sensation of pressure and fulness in the abdomen, intermittent colicky pains and pains in the back, and irregularity of stool. These symptoms may persist for years, but are often of short duration. Increasing disturbances of digestion and occasional colicky pains drive the patient to his physician for aid. The symptoms consist of anorexia, gastric pains, and irregular stools alternately massy and abnormally thin. These patients are often subjected to all manner of treatment intended to relieve the gastric complaints and the inflammation of the large and small intestine, or they are sent to spas, with the result that their condition is aggravated and the digestive symptoms increase. Not until an exact fecal examination has been made is it possible to diagnose the condition, and the method is a rather simple one (see page 125). Deterioration in quality or quantity of the pancreatic trypsin, steapsin or diastase is revealed by the presence of partially digested food remnants in the feces while the patient is taking a test diet. The digestion is more or less seriously impaired, according to whether the entire

pancreas or only part of it is affected. It can be understood that in chronic pancreatitis the digestive disturbances are usually quite pronounced, the entire organ being affected, while tumors of the pancreas, for instance, often cause but slight irregularities, since they destroy only part of the gland. Even occlusion of the pancreatic excretory ducts often causes but slight disturbance so long as vicarious pancreatic function by internal secretion occurs, to say nothing of the fact that the bile and intestinal juice may in part perform the function of the pancreas.

Etiology.—Etiologically, chronic alcoholism, syphilis and arteriosclerosis have to be considered. Chronic lead and mercury intoxications no doubt also play a rôle in the development of chronic pancreatitis. There is no doubt either that chronic pancreatitis may also result from the spreading of inflammatory processes from neighboring organs to the pancreas, as in cholecystitis, cholelithiasis, and gastric and duodenal ulcer. Occlusion of Wirsung's duct by calculi may likewise lead to chronic pancreatitis. Chronic gastritis or achylia may also cause functional disturbances of the pancreatic secretion, it being a well-known fact that the hydrochloric acid of the stomach is a powerful stimulant to pancreatic secretion. These functional derangements may finally eventuate in chronic inflammation of the pancreas.

Pathology.—From an anatomic-pathologic point of view chronic pancreatitis is a cirrhosis of the pancreas with interlobular or intra-lobular connective-tissue proliferation, degeneration of the islands of Langerhans, and atrophy of the parenchyma, the pancreas inclining to contraction and not to enlargement.

Diagnosis.—The test-diet stool method of diagnosis (see Chapter IV) discloses the following digestive disorders in chronic pancreatitis:

Disturbance of Protein Digestion.—In the finely rubbed-up feces one can often detect without the microscope remnants of meat, which are not only characteristic but of diagnostic value on account of the fact that the nuclei are preserved. Schmidt has shown that the cellular nuclei are not digested by the gastric juice, but only by the pancreatic; it is upon this division of proteolytic function that his well-known nucleus test, the demonstration of undigested cellular nuclei in meat particles regained from the feces, is based (see page 126). Kashiwado has simplified this method by repeatedly administering in the middle of the day two colored connective-tissue capsules (especially prepared by Merck), which are later searched for in the stools. Should they be distinctly demonstrable, defective pancreatic function is inferred. There are, however, pancreatic disorders in which the meat digestion is impaired while the nucleus digestion is perfect. If the meat remnants can be detected macroscopically, the microscope will in these cases reveal large numbers of large, angular, muscular fragments, with their

transverse striation well preserved, which, upon close inspection, clearly give the impression of being poorly digested.

Disturbance of Fat Digestion.—In defective steapsin secretion the stool of the test diet contains very large quantities of fat, clearly visible as an abundant fatty layer on the feces finely rubbed up with water, the entire stool looking as if a fatty layer had been poured over it which had congealed after defecation. The influx of bile not being disturbed in pancreatitis, the stool retains its brown color, except that it may at times be lighter in hue than normal; whereas in jaundice it is gray or white owing to interference with the flow of the bile pigment. When the feces are covered with a fat layer, the fat is usually neutral, though it may contain free higher fatty acids. This form of loss of fat, though it points to pancreatic involvement alone, may possibly be due to other causes. Microscopic examination reveals a moderate quantity of fat in the shape of fatty acid needles and soap crystals, and in many instances drops either neutral or acid (see page 120). According to Lohrlich these fat drops can be differentiated by a concentrated solution of Nile blue sulphate, which stains neutral fat red, fatty acid blue.

Disturbance of Starch Digestion.—When a solution of potassium biniodid is added to the feces, more or less numerous undigested starch grains are often discovered. Undigested starch remnants may also be demonstrated in the fermentative processes by applying the incubator test (see page 121). If the digestive disturbance, as described, has persisted for a long time or is of a very severe nature, it will sooner or later lead to catarrh of the small and large intestine, demonstrable in the fecal examination by the presence of mucus or putrefactive processes. Should there be simultaneous achylic or subacid gastric conditions, the feces will contain undigested remnants of connective tissue.

If, in addition to these digestive disorders, the anamnesis and other clinical findings (pain on pressure in the pancreatic region, colicky pains, emaciation) are taken into consideration, there should be no difficulty in arriving at a diagnosis of pancreatic affection. Simultaneous diabetes would, of course, strengthen the diagnosis still further. Demonstration of trypsin and diastase (see pages 125-126) in the feces may in some cases be omitted, for should the tests already described prove negative the digestive disturbances are often quite characteristic enough to clear up the diagnosis. It is, of course, not always possible to diagnose at once chronic pancreatitis to the exclusion of other affections of the pancreas, further observations being frequently necessary for this purpose. Chronic pancreatitis should be particularly suspected when the disease remains stationary or undergoes temporary improvement.

Since the contents of the duodenum can be easily removed by the duodenal tube (see page 98), the condition of the pancreas can in

some measure be estimated by this means. The presence of bile and pancreatic secretion in the duodenum permits gauging the pancreatic function. The presence of all three ferments in sufficient quantity indicates normal activity of the pancreas. If one of the ferments is constantly absent, this usually indicates chronic pancreatitis (Einhorn). A tumor of the pancreas may exist notwithstanding the presence of all three ferments.

Oil Test Breakfast. It has been found that oil introduced into the stomach usually induces regurgitation of pancreatic juice, bile and succus entericus into the stomach. Next to the removal of the intestinal juices by the duodenal tube and the duodenal bucket, for demonstrating the presence or absence of the pancreatic ferments, the oil test breakfast is best for analytic purposes. This test depends upon the splitting of a neutral oil by the pancreatic ferment into fatty acid and glycerin. The fatty acids are recognized by their green color, due to the formation of copper salts. Palmin is the oil that should be used. The patient is to take an oil test breakfast on an empty stomach—30 grams (one ounce) of rice starch dissolved in $\frac{1}{2}$ liter (eight ounces) of warm water (with a little salt if desired), to which is added 75 Cc. (2½ ounces) of warm palmin oil; mix thoroughly. The whole quantity is taken at once. In two or two and a half hours the stomach contents should be removed through a tube and tested immediately. In order to secure the reaction two solutions are necessary. The first consists of petroleum ether ninety parts and distilled water ten parts. The second consists of a 3-per-cent. solution of copper acetate in distilled water. Equal parts of the first solution and the stomach contents are placed in a test tube, thoroughly shaken, and allowed to stand; then the supernatant ether is poured into another test tube, to which an equal quantity of the second solution is added, and the tube shaken. If there are any fatty acids present, they unite with the copper, forming a salt with an intense green color, easily discernible to the eye. If there is no pancreatic juice present, the solution remains clear, showing that there has been no splitting of the palmin.

Loewi's Pupillary Reaction.—We owe to Loewi (1908) the discovery that epinephrin will dilate the pupil in animals from which the pancreas has been removed. From this fact it has been inferred that pupillary reaction to epinephrin indicates insufficiency of pancreatic secretion—Graves' disease being excluded. The pupillary test is made simply by instilling a few drops of epinephrin solution, 1:1000, in the conjunctival sac, and noting the effect.

Cambridge Reaction.—In his recent investigations on the urine of patients suffering from pancreatic disease, Cambridge has found that the appearance of sugar in the urine in diseases of the pancreas is preceded by a marked rise in the excretion of dextrin, which falls when glycosuria is established. He has elaborated a test known as the Cambridge reaction. The reaction depends upon the

formation of an osazone by the pentose (xylose) split off from the dextrin by hydrolyzing with hydrochloric acid; and in the quantitative method the "iodin coefficient" of the urine is a measure of its content of dextrin. The determinations of the "iodin coefficient" are not only useful in the diagnosis of diseases of the pancreas, but also afford a means of detecting the preglycosuric stage of diabetes.

Pain and tenderness on pressure in the vicinity of the twelfth dorsal and first and second lumbar vertebrae is significant of disease of the pancreas.

The presence of all the ferments in sufficient quantity indicates normal pancreatic activity. This can be easily ascertained by an examination of the duodenal contents (see page 103).

Prognosis.—The prognosis of chronic pancreatitis is uncertain, but not entirely unfavorable, since, with a correct mode of living, the affection may either come to a standstill or show improvement.

Treatment.—The preceding explanations indicate the logical internal treatment. The first point is to modify or remove the cause. Biliary calculi, cholecystitis, gastric or duodenal ulcer, if present, should be most thoroughly treated, because a cure of these conditions affords substantial hope of arresting the pancreatic affection. A stay at Saratoga, Carlsbad or Marienbad has often a favorable effect. Prolonged rest in bed, in conjunction with a carefully regulated diet, may likewise exert a favorable influence upon the pancreatic affection. Any affection of the stomach, in particular achylia or subacidity, atony or chronic catarrhal affection, should be carefully treated, especially when it has led to gastrogenic diarrhea (see page 673). Irrigation of the stomach is often very useful in atony of that organ. In subacid and achylic conditions, hydrochloric acid and pepsin should be administered in abundant quantities to relieve the gastrogenic diarrhea (see page 258).

Small quantities of hydrochloric acid may also be taken before meals in order to incite pancreatic secretion.

Regulation of the diet is of the greatest importance. Generally speaking, it is easy to prescribe the necessary diet, based upon the findings of the fecal examinations. It should fulfil the following requirements:

1. *Limitation of Fat.*—Butter, cream, oil, and meat fat should be given as sparingly as possible. Milk is allowable in small quantities only.

2. *Limitation of Protein.*—Meat and eggs should be limited to small quantities, the meats being thoroughly chopped and all egg dishes prepared in the most readily digestible form. Smoked and pickled meats are prohibited. Rare meat is permissible only in the shape of carefully scraped beef or tender ham. Vegetable proteins (roborat, mutase, etc.) may be administered as substitutes

for animal food. Gelatins supply but little putrefactive material, and are therefore valuable. Erepton, consisting of meat artificially digested by the action of trypsin and erepsin, and having a nitrogenous content of about 12 per cent., is recommended.

3. *Carbohydrates* may be given in abundant quantities, preferably in the shape of fine farinaceous food, or soup prepared with fine soup flour. White bread, biscuits, cakes, rice, hominy, sago, dry vegetables, mashed potatoes, etc., are, of course, likewise included in the diet. Individualizing is a necessary requisite, and the menu should not be too monotonous. With due regard to the severity of the digestive disturbance, green vegetable purée (spinach, etc.), apple sauce, and similar dishes may be allowed. If diabetes, at any rate severe diabetes, is present, saccharin should be substituted for sugar. Beverages containing carbon dioxide, such as beer, are best avoided, but wine is allowable in small quantities.

To assist the digestion of fat as well as of meat, pancreatin and pankreon are administered with advantage (see page 262). Pancreatin is effective in a neutral or weakly alkaline solution only, and is therefore particularly useful in cases of achylia gastrica. In normal gastric conditions it should be administered with bicarbonate of sodium, a quarter of an hour after meals. Pancreatin preparations in most cases very materially aid in the splitting up and absorption of fat as well as the digestion of starches. They may be continued for a long time. The carbohydrate digestion may also be assisted by vegetable diastase—for example, taka-diastase, administered during meals in doses of 0.2 to 0.5 Gm. (3 to 7½ grains).

Slow eating, regular distribution of the meals over the day, and rest after meals are of course necessary. Warm compresses, dry or moist, applied to the abdomen after meals, usually produce a comfortable sensation. Light cases of chronic pancreatitis do not contra-indicate careful electric treatment, abdominal massage, or baths and hydrotherapeutic measures in a mild form.

The above exhausts the internal therapeutic possibilities in chronic pancreatitis, according to our present knowledge. Functional disturbances of the pancreatic secretion (achylia pancreatica) are, of course, to be treated in the same way.

Surgical Treatment.—Surgery is generally powerless in chronic pancreatitis. It might possibly come in for consideration in chronic peripancreatic processes for the resection of adhesions, splitting the pancreatic capsule, etc., but it is very difficult to diagnose peripancreatitis with that degree of certainty which is necessary in advising operation.

Acute Pancreatitis.—Acute inflammation of the pancreas is nearly always due to bacteria, principally the *Bacterium coli communis*, invading the pancreatic duct from the intestine. There may also be hematogenous bacterial invasion of the pancreas, notably in the course of infectious diseases, as in pyemia. Strepto-

cocci, staphylococci, typhoid bacilli and Fränkel's pneumococci have all been found in these infections. Acute pancreatitis also appears as a sequel to affections of the liver and bile ducts, gastric and duodenal ulcers, and carcinoma of the stomach, and occasionally in association with pancreatic calculi. It is more often observed in men than in women. The inflamed pancreas is usually enlarged and very plethoric. The inflammation, when very intense, may lead to hemorrhages into the pancreas (pancreatitis hemorrhagica). Suppuration of the pancreas may also occasionally occur (pancreatic abscess). Acute pancreatitis usually begins with violent pains in the upper abdominal region, accompanied by vomiting and collapse, and often by symptoms of ileus and peritonitis. The body temperature is often elevated. Fitz's syndrome, suggesting acute pancreatitis, consists of severe epigastric pain, vomiting and collapse, appearing suddenly and followed within twenty-four hours by a circumscribed swelling in the epigastrium, with tympanites. A percussion sign of acute pancreatitis "so absolute," according to its discoverer, "that it can be felt," is described by Todd, of Adelaide, Australia, as "dulness in both flanks, unaltered by any change of posture," and attributable to a collection of blood in both kidney pelves. A general or local cyanosis is a common symptom. The test-diet stool is characteristic. The prognosis is very serious, the affection, as a rule, running a fatal course.

Treatment.—The treatment is surgical, consisting in laparotomy, removal of the pathologic glandular tissue, and evacuation of the pus. Professor Gosset, of Paris, believes the gall bladder should be drained in every case.

Internal Treatment in Other Affections of the Pancreas. Nothing is available here but regulation of the diet. This, however, is by no means an unimportant point, and may be attended with very good results in some of these affections. Dietetic treatment is based upon experimental physiology. Pawlow's experiments upon dogs and the observations of Wohlgemuth upon pancreatic fistule in man have shown that the pancreatic secretion, aside from psychic influences, can be characteristically affected by various articles of food. It has been shown that carbohydrates (potatoes, bread, farinaceous food, etc.) incite pancreatic secretion to a considerable extent. Protein food is much less effective in this respect, while pancreatic secretion is almost completely arrested after the ingestion of fat. It is also a well-known fact, as already stated, that hydrochloric acid stimulates pancreatic secretion, while bicarbonate of sodium distinctly inhibits it. These effects upon pancreatic function are not all direct; we know that proteins and carbohydrates do not of themselves directly influence pancreatic secretion; on the other hand, fats have a moderately stimulating effect. These various foods influence the pancreas through the stomach, by their effect upon the secretion of free hydrochloric acid. Con-

for animal food. Gelatins supply but little putrefactive material, and are therefore valuable. Erepton, consisting of meat artificially digested by the action of trypsin and erepsin, and having a nitrogenous content of about 12 per cent., is recommended.

3. *Carbohydrates* may be given in abundant quantities, preferably in the shape of fine farinaceous food, or soup prepared with fine soup flour. White bread, biscuits, cakes, rice, hominy, sago, dry vegetables, mashed potatoes, etc., are, of course, likewise included in the diet. Individualizing is a necessary requisite, and the menu should not be too monotonous. With due regard to the severity of the digestive disturbance, green vegetable purée (spinach, etc.), apple sauce, and similar dishes may be allowed. If diabetes, at any rate severe diabetes, is present, saccharin should be substituted for sugar. Beverages containing carbon dioxid, such as beer, are best avoided, but wine is allowable in small quantities.

To assist the digestion of fat as well as of meat, pancreatin and pankreon are administered with advantage (see page 262). Pancreatin is effective in a neutral or weakly alkaline solution only, and is therefore particularly useful in cases of achylia gastrica. In normal gastric conditions it should be administered with bicarbonate of sodium, a quarter of an hour after meals. Pancreatin preparations in most cases very materially aid in the splitting up and absorption of fat as well as the digestion of starches. They may be continued for a long time. The carbohydrate digestion may also be assisted by vegetable diastase—for example, taka-diastase, administered during meals in doses of 0.2 to 0.5 Gm. (3 to 7½ grains).

Slow eating, regular distribution of the meals over the day, and rest after meals are of course necessary. Warm compresses, dry or moist, applied to the abdomen after meals, usually produce a comfortable sensation. Light cases of chronic pancreatitis do not contra-indicate careful electric treatment, abdominal massage, or baths and hydrotherapeutic measures in a mild form.

The above exhausts the internal therapeutic possibilities in chronic pancreatitis, according to our present knowledge. Functional disturbances of the pancreatic secretion (achylia pancreatica) are, of course, to be treated in the same way.

Surgical Treatment.—Surgery is generally powerless in chronic pancreatitis. It might possibly come in for consideration in chronic peripancreatic processes for the resection of adhesions, splitting the pancreatic capsule, etc., but it is very difficult to diagnose peripancreatitis with that degree of certainty which is necessary in advising operation.

Acute Pancreatitis.—Acute inflammation of the pancreas is nearly always due to bacteria, principally the *Bacterium coli communis*, invading the pancreatic duct from the intestine. There may also be hematogenous bacterial invasion of the pancreas, notably in the course of infectious diseases, as in pyemia. Strepto-

cocci, staphylococci, typhoid bacilli and Fränkel's pneumococci have all been found in these infections. Acute pancreatitis also appears as a sequel to affections of the liver and bile ducts, gastric and duodenal ulcers, and carcinoma of the stomach, and occasionally in association with pancreatic calculi. It is more often observed in men than in women. The inflamed pancreas is usually enlarged and very plethoric. The inflammation, when very intense, may lead to hemorrhages into the pancreas (pancreatitis hemorrhagica). Suppuration of the pancreas may also occasionally occur (pancreatic abscess). Acute pancreatitis usually begins with violent pains in the upper abdominal region, accompanied by vomiting and collapse, and often by symptoms of ileus and peritonitis. The body temperature is often elevated. Fitz's syndrome, suggesting acute pancreatitis, consists of severe epigastric pain, vomiting and collapse, appearing suddenly and followed within twenty-four hours by a circumscribed swelling in the epigastrium, with tympanites. A percussion sign of acute pancreatitis "so absolute," according to its discoverer, "that it can be felt," is described by Todd, of Adelaide, Australia, as "dulness in both flanks, unaltered by any change of posture," and attributable to a collection of blood in both kidney pelves. A general or local cyanosis is a common symptom. The test-diet stool is characteristic. The prognosis is very serious, the affection, as a rule, running a fatal course.

Treatment. The treatment is surgical, consisting in laparotomy, removal of the pathologic glandular tissue, and evacuation of the pus. Professor Gosset, of Paris, believes the gall bladder should be drained in every case.

Internal Treatment in Other Affections of the Pancreas.—Nothing is available here but regulation of the diet. This, however, is by no means an unimportant point, and may be attended with very good results in some of these affections. Dietetic treatment is based upon experimental physiology. Pawlow's experiments upon dogs and the observations of Wohlgemuth upon pancreatic fistulæ in man have shown that the pancreatic secretion, aside from psychic influences, can be characteristically affected by various articles of food. It has been shown that carbohydrates (potatoes, bread, farinaceous food, etc.) incite pancreatic secretion to a considerable extent. Protein food is much less effective in this respect, while pancreatic secretion is almost completely arrested after the ingestion of fat. It is also a well-known fact, as already stated, that hydrochloric acid stimulates pancreatic secretion, while bicarbonate of sodium distinctly inhibits it. These effects upon pancreatic function are not all direct; we know that proteins and carbohydrates do not of themselves directly influence pancreatic secretion; on the other hand, fats have a moderately stimulating effect. These various foods influence the pancreas through the stomach, by their effect upon the secretion of free hydrochloric acid. Con-

may succumb to the first attack. In favorable cases the acute stage may be overcome, with the result that a latent stage develops, the necrotic areas becoming encapsulated. The pancreas is at first considerably enlarged, dark red, and soft; but as the affection continues it becomes hard and firm, distinctly demarcated against its surroundings. Finally it is reduced to necrotic pulp inside its own capsule, or it continues to become harder and firmer until it lies as a dead organ in an abscess cavity. Should the necrosis spread beyond the pancreas, and active pancreatic juice invade the abdominal cavity, a so-called fatty tissue necrosis will result. Whenever the pancreatic juice comes in contact with fat, decomposition of the neutral particles in the fat cells will result. Throughout the pancreas and adjacent region are found yellowish-white points, from a size just discernible to the size of a pinhead, indicating the location of the chemically decomposed fat.

The treatment consists in opening the abscess cavity containing the necrotic tissue. The prognosis is very bad.

PANCREATIC CYSTS.

There are to be distinguished:

(1) *Genuine cysts*, hollow spaces invested with epithelium, resulting from proliferation of the glandular ducts and the acini of the pancreas, from stagnation in the flow of the pancreatic juice, or from degeneration of the pancreatic tissue, as, for instance, after infectious diseases.

(2) *Cystoid structures* without any epithelial lining, which may result from softening of the enlarged pancreas, necrosis of some of its parts, and blood effusions. The more intense the pathologic processes which destroy the pancreatic tissue, the more rapidly the cysts develop. The comparative rapidity of cyst development therefore admits of a conclusion in regard to the original affection of the pancreas, an important point in the anamnesis. Gradually the cyst, owing to its increasing size, exerts an unpleasant effect by pressure upon the neighboring organs, especially the stomach and the transverse colon. In any case the diagnosis is by no means an easy matter. The Roentgen ray and bismuth may assist in the diagnosis by showing a uniform curvature on the left of the stomach, due to pressure of the cyst.

Treatment.—The only remedy is laparotomy, followed by suturing the cyst into the laparotomy wound, with unilateral or bilateral opening. The cyst having been opened, there remains its gradual obliteration by the measures employed in the obliteration of fistule (see page 629), particularly Wohlgenuth's diet.

TUMORS OF THE PANCREAS.

The pancreas is subject to primary sarcoma and carcinoma. The latter is the more frequent and may involve the whole gland, the head or the tail, or the duct of Vater. Permanent or periodic pains and colic, jaundice and early cachexia point to carcinoma. Oftentimes the tumor cannot be palpated, owing to sensitiveness and to the ascites which is present in most cases. Carcinoma of the head of the pancreas induces progressive jaundice. A common cause of symptomless jaundice is malignant disease of the pancreas (see page 614).

Treatment.—A radical operation is impossible in most cases. When the pylorus is occluded, some relief may be afforded by gastroenterostomy.

PANCREATIC CALCULI.

Pancreatic calculi are due to some antecedent defect in pancreatic function. The substances of which the calculi are composed (lime salts, cholesterol) are in solution in normal pancreatic juice, but are precipitated when qualitative changes in the juice take place. Changes of this kind may occur as a result of bacterial invasion from the intestine, occlusion of the excretory ducts from stricture, or compression from without. Chronic pancreatitis likewise plays a rôle in the etiology of this affection. The catarrhal secretion in the ducts and the desquamated epithelium form the nucleus around which the calculi are built up. The latter float in the current of the secretion toward the excretory ducts of the gland to the papilla, where they meet with resistance; if this resistance is not overcome, stagnation and decomposition of the secretion results, leading in many cases to the formation of new stones behind the original one. When there is a complicating infection, suppurative pancreatitis may easily supervene, likewise necrosis. In the absence of any infection there is a tendency to chronic changes, chronic pancreatitis, and cyst formation.

Among the clinical manifestations are colic, swelling of the gland (which is sometimes palpable), and expulsion of stones (which must be carefully looked for in the feces when pancreatic calculi are suspected). Should larger stones be formed, leading to stagnation of the secretion and chronic pancreatic changes, there will be the same digestive disturbances as are observed in chronic pancreatitis.

Treatment.—The internal treatment has already been discussed (see page 629). If the diagnosis of pancreatic calculi is in all probability correct, surgical treatment may be instituted.

PANCREATIC INFANTILISM.

This rare condition is due to abnormal inhibition of pancreatic secretion. It is characterized by lack of bodily and sexual development, and as a result of the arrest of pancreatic secretion a condition of chronic enteritis ensues which is manifested by a constant diarrhea associated with flatulence. The retarded bodily development and the chronic diarrhea are the main clues to the diagnosis.

This condition can be completely cured by the administration of pancreatic extract.

CHAPTER XXXIV.

ACUTE ENTEROCOLITIS.

ACUTE INTESTINAL CATARRH ACUTE GASTROENTERITIS—ACUTE COLITIS CHOLERA MORBUS—CHOLERA NOSTRAS—ACUTE DIARRHŒA.

PRIMARY acute intestinal catarrh, like acute gastric catarrh, is a disease of quite frequent occurrence.

Etiology.—Étiologically, four varieties of acute intestinal catarrh are recognized:

(1) *Acute Infectious Catarrh.*—This form of enteritis is of bacterial origin. The *Bacillus coli communis* and the *Bacillus enteritis* apparently play an important rôle in the etiology. Numerous other species of bacteria are, however, frequently found, but these have usually been introduced with some article of the diet. It also seems probable that acute infectious catarrh may be induced by the agency of numerous entozoa and endamebæ.

The various forms of colitis which occur in cases of chronic constipation, due to stagnation and bacterial decomposition of fecal matter (colitis stercoralis), belong under the general classification of infectious catarrh.

(2) *Alimentary Catarrh.*—Acute intestinal catarrh may be caused by the ingestion of food that has not undergone decomposition. Wholesome articles of diet, improperly prepared, insufficiently masticated, and ingested in excessive quantities, can cause it. Usually, however, it is due to food or drink (water, milk, sausage, fish, butter, ice, fruit) which contains toxic substances (ptomaines). The warm season of the year, and tropical climates, by favoring the decomposition of foodstuffs, contribute to the prevalence of intestinal catarrhs.

(3) *Catarrh Due to Exposure to Cold.*—There can be no doubt that sudden cooling of the abdomen, the back, or the feet (wet feet), and cold beverages, may induce acute intestinal catarrh.

(4) *Catarrh from Intoxication.*—This condition may be due to either medicaments or poisons. Among drugs that produce catarrh when taken for a considerable length of time are preparations of mercury (even when injected intramuscularly), arsenic, phosphorus, the emetics, drastic purgatives, turpentine, copaiba, etc. The poisons chiefly to be considered are the acids, alkalis, and alcohol.

Pathology.—Acute catarrh of the intestine is characterized by swelling and relaxation of the mucous membrane. According to

the intensity of the disease, the mucous membrane is reddened in a circumscribed area or for its entire length, in consequence of hyperemia. The reddening may advance so far as to become a dark purple discoloration. The solitary follicles are usually swollen, though Peyer's patches do not seem to be involved. The mucous membrane is more or less covered with mucus, which may appear clear like glass, cloudy, or stained with blood. Small superficial abrasions of the epithelium are of common occurrence. The contents of the intestine are usually fluid. Microscopically there may be recognized distended bloodvessels of the mucous membrane and occasionally minute extravasations of blood. The interspaces between the crypts of Lieberkühn are generally dilated and infiltrated with round cells. The epithelium of the glandular cells is frequently opaque. The glands themselves are often markedly enlarged, relaxed, and elevated above the surface, in consequence of infiltration of the connective tissue.

Acute intestinal catarrh is most frequently localized in the large intestine (colitis) exclusively. In many cases, however, the small intestine is simultaneously affected; this condition is called mixed catarrh. The noxious agencies above named induce primarily an acute gastritis, and from the stomach the inflammation extends to the intestine, thus forming the well-known picture of acute gastroenteritis. Disease of the small intestine only is a very rare affection, although, according to Nothnagel, it undoubtedly does occasionally occur. An isolated acute catarrhal disease of the duodenum is not rare, but the disease is usually associated with acute gastritis (gastroduodenitis).

Symptoms.—Among the first symptoms of acute intestinal catarrh are abdominal discomfort and pain, and loss of appetite. The characteristic diarrhea develops early, the number of the stools being proportional to the gravity of the affection. The evacuations become more liquid as the frequency of the discharges increases, and the abdominal pains tend to become more severe during this period. With coincident acute gastritis the symptoms of this condition may lead, perhaps overshadow, those of the intestinal catarrh. But even without the presence of gastritis there may supervene great discomfort, nausea, pressure in the epigastric region, and occasionally vomiting. The general subjective condition depends upon the intensity of the catarrh; in mild cases it is not affected at all. Moderately severe cases are usually attended with lassitude and sensations of weakness. Severe cases with profuse diarrhea may induce an alarming condition of weakness, especially in young patients and those well advanced in years. Fever is usually absent in the lighter and moderately severe cases; occasionally, however, there may be high fever. Enlargement of the spleen is rare. Albumin and casts are frequently found in the urine of cases of severe catarrh, particularly in patients advanced

in years. The urine soon returns to a normal condition with the recession of the catarrh.

Examination of the abdomen will occasionally elicit a moderate diffuse tenderness to pressure, which becomes pain when the colon is involved. Frequent borborygmi and other adventitious sounds are heard. When the abdominal walls are thin it is sometimes possible to observe visually the acceleration of the peristaltic movements.

Diagnosis.—The deciding points in establishing the diagnosis of acute intestinal catarrh, and for the localization of the disease in the large or small intestine or both, are ascertained by the condition of the alvine discharges. The more or less pronounced liquidity of the feces and the amount of mucus present are characteristic points. The fecal matter of acute catarrh of the large intestine is usually of a dark brown color and offensive odor, and there may be discerned isolated particles of mucus; it contains easily visible mucous-looking flocculi, transparent or turbid, and shreds which are occasionally tinged with blood, especially in cases in which the inflammation has gained in intensity. In cases of catarrh of the small intestine, which are not so frequent, the stools are lighter in color. When the catarrh is of great intensity the evacuations may be actually green, this color being caused by the presence of unaltered bile pigment. There may be much mucus, in small lumps, intimately mixed with the feces, imparting to the stools a jelly-like consistency. If the feces contain undigested remnants of food, such as meat, starch, and fat, it is probable that the small intestine is inflamed. Fecal particles (mucous flocculi, fatty residues, or muscle fibers) that become green on the addition of a 5-per-cent. solution of sublimate (Schmidt's sublimate test—page 116) furnish thereby proof of the presence of unchanged bile pigment. Dark, mucoid, offensive stools, containing undigested particles of food, indicate the simultaneous existence of catarrh of both the small and the large intestine.

For purposes of treatment it is only necessary to distinguish between these two sections of the intestinal canal in localizing the catarrh; it is scarcely possible to fix exactly the particular region affected by the catarrhal process. The fact that the duodenum is sometimes the seat cannot be doubted, and this condition is usually found associated with acute gastritis. It may be diagnosed if jaundice (catarrhal icterus) supervenes in acute gastritis. It is not necessarily accompanied by diarrhea. In cases of duodenitis, examination of the duodenal contents after removal with the duodenal tube shows stringy mucus, Gram-positive motile bacilli, and numerous cocci.

It is sometimes difficult to differentiate between an acute grave colitis and a mild acute dysentery. It is also occasionally difficult to distinguish, during the first days of the disease, between

acute intestinal catarrh with a temperature and a case of beginning typhoid fever.

Prognosis. The course of acute intestinal catarrh is usually favorable. With proper care complete recovery may be expected in three or four days, often sooner. In a few cases, however, there remains a certain hypersensitiveness of the intestine, and a tendency to recurrence of the catarrh after slight dietary errors. Severe catarrh of the large intestine, of several weeks' duration, may occasionally be very slow in healing; some cases may indeed resist all treatment and become chronic. Acute enteritis or gastro-enteritis rarely runs a serious course; but profuse diarrhea may supervene, evidently under the influence of particularly virulent or toxic microorganisms—a view that is confirmed by the frequent occurrence of this grave form of disease during the summer. The evacuations finally consist of serous exudate with mucous and fibrinous flocculi thoroughly mixed with pus and epithelium; they are whitish-gray, resembling rice-water, of alkaline reaction, and may occur as often as twenty to thirty times a day. Violent vomiting also may be present. The quantity of urine becomes much less in consequence of the great loss of water, and anuresis may supervene. Albumin and casts are frequently found in the urine. This intense catarrhal disease of the intestine is frequently sufficient to completely prostrate even vigorous persons. It is a direct menace to life in the case of old people and children. There may finally occur, in a most sudden manner, cramps of the calves of the legs, hoarseness, coldness of the extremities, and cardiac collapse. Because of the similarity of the stools to those of Asiatic cholera patients, and the resemblance of the clinical symptoms to those of genuine cholera, this affection has been termed *cholera nostras*, or *cholera morbus*. Notwithstanding the gravity of the symptoms, the disease, when properly treated, usually runs a favorable course.

Treatment.—Persons subject to acute intestinal catarrh, as taught by their own experience, should as a matter of prophylaxis guard against colds and against errors in diet. They should be advised to clothe themselves warmly, and to wear in winter a warm flannel or woollen abdominal bandage or warm undergarments. They should avoid cold beverages and foods, and coarse articles of diet such as fresh fruit, radishes, cucumbers, and salads. When the physician is called to treat a case of acute catarrh of the intestine, the cause of the attack must be considered first. If it can be ascertained that harmful foods or infectious substances have been introduced into the stomach, the indication is, of course, to empty the intestine as rapidly as possible. Nature, indeed, assists herself in these cases, for the decomposition products accumulated in the gut stimulate peristaltic movements, inducing diarrhea, by which the harmful materials are expelled. It is frequently observed that improvement begins after a number of

copious diarrhetic discharges; the bowels themselves have acted upon the etiologic indication. In many cases, however, the spontaneous evacuation of the intestine is not sufficient, and notwithstanding the free movement no improvement takes place. In such cases nature must be assisted, that the elimination of the noxious material may be more thorough. Castor oil and calomel serve this purpose. The former may be given in tablespoonful doses, one tablespoonful every hour until effective. Because of its disagreeable taste it is often administered in gelatin capsules or in emulsions.

The *National Formulary* gives the formula of emulsion olei ricini, which contains one-third of its volume of castor oil. Three tablespoonfuls is the average dose.

Calomel acts safely, rapidly, and mildly, without causing inflammatory irritation of the bowel. It is given in doses of 0.02 to 0.05 Gm. ($\frac{1}{4}$ to 1 grain), two or three such powders at intervals of two hours.

	Gm. or Co.	
R—Hydrargyri chloridi mitis	0 3	gr viiss
Sodu bicarbonatis	1 0	gr. xv
Misce et ft. chart. no. viii.		
Sig.—One every hour, followed by a Scidlitz powder.		

	Gm. or Co.	
R—Hydrargyri chloridi mitis	0 3	gr. v
Sacchari albi	0 6	gr. x
Misce et ft. chart. no. i.		

Sig.—Take at once, to be followed in two hours by a tablespoonful of Rochelle salt.

Instead of these purgatives, any of the well-known bitter mineral waters (see page 254) may be given. When the stomach is involved the bowel should be emptied by means of enemata.

The evacuation of the bowel having been accomplished, attention must be given to the care of the stomach. In many cases treatment commences without previous evacuation of the bowels, no toxic or irritating material being present, or the case being one of catarrh due to a "cold." The care of the stomach is best secured by rest in bed, the duration of which will depend on the severity of the attack. Some slight cases may be permitted to get up and around as soon as the second day, but in obstinate cases rest in bed for several days is indicated. While in bed, hot cloths should be applied to the abdomen, and frequently changed. Dry or moist applications may be made by means of the "Priessnitz" bandage (see page 250).

The diet is of great importance in the avoidance of irritation. It is better not to allow any food to be taken during the first twenty-four hours, that the decomposition processes in the intestine may receive no encouragement. Therefore, on the first day, it is well to prescribe complete abstinence, or only an apparent nutrition consisting of thin water broths, thin meat broth (mutton broth),

tea with cognac, white-of-egg water consisting of one white of egg to 200 Cc. (7 ounces) of water with the addition of a little table salt and cognac. This pseudonutrition suffices for the first two days, and in grave cases with violent diarrhea and colic it may be continued for longer periods. During the first days it is useful to administer carminative teas, valerian, peppermint, thyme, fennel, or anise-seed. Under this treatment the symptoms usually improve within one or two days. A somewhat more copious diet may then be allowed, proceeding with great care. The food should be free from liability to decomposition, as porridges (thoroughly cooked and strained) either of oat or barley meal, tea with a little milk or claret, cocoa, acorn-cocoa, blackberry decoctions, whortleberry jelly or wine, and soups with rice, tapioca, leguminous flours and infant-food flours. Sugar should be replaced in the drinks by saccharin. Should there be much thirst, milk of almonds may be given; but always freshly prepared, for it decomposes readily; it has a slightly astringent effect.

Watching and controlling the bowels carefully, the physician may gradually allow a more varied diet—bouillon with egg, water-soaked stale bread or toast, boiled water with claret, soups with ground-up stale bread and sweetbread, or rice and hominy. This diet should be persevered in until the bowels have become completely quiescent, the evacuations firm, and all abdominal pains and sensitiveness to pressure have disappeared. Only now more solid food may be taken, such as scraped meat free of connective tissue, squab, chicken, scraped white meat, mashed potatoes, and vegetables ground fine and passed through a sieve, such as peas, lentils, spinach; with cocoa or chocolate. The course of the disease being normal, a full diet is soon assimilable. For some weeks, however, foods rich in cellulose should be avoided, such as radishes, carrots, cabbage and fresh fruit, as well as fatty and sour substances. Milk is to be avoided during the first days of an acute intestinal catarrh, because of the fact that in occasional instances, which cannot be predetermined, it induces diarrhea. After the patient has begun to partake of potatoes or other specially prepared solid food, milk may be permitted, either boiled by itself or together with cocoa or chocolate. The astringent effect of three-day kefir seems to be advantageous in acute intestinal catarrh.

It is impossible to lay down a diet for all cases that will be non-irritating and easily assimilable. So far as milk is concerned, although it often proves exceedingly useful when given in small quantities, yet in the majority of cases of catarrh of the small intestine it had best not be given. Experience has shown that if the small intestine is put at rest for a short time it is possible to give milk later and with good result.

Disease of the large intestine, even though it may be advanced, does not contra-indicate the use of milk; on the contrary, milk,

especially when it is the only food taken, is exceedingly useful. In achylia gastrica and in hyperchlorhydria, milk as a diet occupies first place, but it should be omitted when there is an associated catarrh of the small intestine.

To obtain a complete cure it is absolutely essential to persevere with the non-irritating diet for as long a time as possible. In this respect one should be rather too rigorous than lenient. Should the patient lose flesh rather rapidly during the first few days, one should not be misled and permit the ingestion of larger quantities of food in order to prevent further loss of weight. The keeping of the affected intestine at rest is the paramount consideration. Upon recovery the patient quickly regains all he may have lost in weight.

Nothnagel has called our attention to the following truths regarding the treatment of catarrhal inflammation of the mucous membrane of the intestine: (1) A genuine cure of an acute catarrh can only be brought about through the regenerating processes going on in the affected tissues. (2) Complete recovery is possible in the acute form of intestinal catarrh only. The second point is especially emphasized, and the assertion is made that chronic intestinal catarrh is difficult to cure. It is therefore of the utmost importance to prevent the acute catarrh from becoming chronic. This can be accomplished not so much by medication as by a well regulated regimen. A constipating diet (see page 172) is essential.

In patients of advanced years suffering from severe intestinal catarrh and great weakness of the heart, it is proper to allow the administration of alcohol, in the shape of punch, tea with claret, or cognac and water.

Should the small intestine be the principal seat of the disease it may occasionally be necessary to greatly reduce the ingestion of water. The water impoverishment in such cases may well be met by the use of the Murphy drip (see page 239).

Medicaments, generally speaking, do not play an essential part in the treatment of acute intestinal catarrh. In cases with obstinate diarrhea, violent abdominal pains, and vigorous peristalsis, opium (see page 274) will be chiefly relied upon, but its employment in the treatment of young children and the aged demands great care. In cases of persistent vomiting, opium may be administered in the form of suppositories. Opium, however, should only be made use of for a few days. Should constipation continue several days following the use of opium, it may be safely and unhesitatingly left to itself; but if it should continue too long, or if it should occur even without the taking of opium a rather frequent occurrence—purgatives should never be employed; instead of them, enemata of soap-water or oil should be given (see page 223).

In excessive peristalsis of the intestine, as in diarrhea and dysentery, benzyl benzoate is often of great advantage. In doses of 2

Cc. (30 minims) in water three or more times daily it promptly checks the diarrhea (see page 276).

Astringents, as a rule, are not employed in acute catarrh, unless it be of remarkably long duration. For children and old people who must not be given opium, subgallate of bismuth may be administered—for adults, 0.5 to 1 Gm. (8 to 15 grains) as powder several times daily; for children it may be given in chalk mixture:

	Gm. or Cc.	
R—Bismuthi subgallatis	2 0	℥ss
Mistura cretae	q. s. ad 60.0	℥ij
Misce		
Sig = Teaspoonful every one or two hours.		

Bolus alba triturated in water has recently been recommended, in doses of 60 to 100 Gm. (℥ ii-ij) once a day, before breakfast. We may occasionally cut short cases of acute enteritis without any other treatment. Bolus alba, or kaolin, is commonly known by the name of porcelain clay. It is used as an absorbent powder, dusted on the surface of the body in irritable skin conditions. It is not easily affected by chemical reagents. Such large quantities as above specified are rather disagreeable to take.

Cardiac tonics, such as digitalis, callein, strophanthus, camphor and alcohol, are to be given from the commencement of the treatment to old people with weak heart.

Cases of enteritis caused by acids, alkalis and poisons are to be treated as stated under the heading Toxic Gastritis, and by the appropriate antidotes.

Treatment of Cholera Morbus.—Besides the acute intestinal symptoms to be treated, as above mentioned, it is necessary to employ most energetic measures in combating the grave general symptoms, such as the loss of water from the body and the collapse. Such patients have to be kept very warm, with hot bottles to the feet, the bed heated, hot fomentations to the abdomen, and vigorous rubbing of the extremities. Stimulants are to be freely given in the form of alcoholic drinks, such as punch, and hot tea with claret or brandy. When the heart is weak, camphor, ether, caffenin or digitalone may be given subcutaneously.

In acute gastroenteritis of infants, sea-water plasma has been found very efficacious. It has been used in grave cases with good results. The use of sea-water in such cases is never contra-indicated. Rapid recoveries occur in babies when milk, broths, and even boiled water are vomited. Not only does vomiting cease after two of three injections, but the diarrhea also stops. In cases of extreme malnutrition associated with acute gastroenteritis the child can frequently take milk after the first injection. In Paris there are special places called "Sea-water Dispensaries for Poor People" where the children are brought for the injection of the sea-water plasma. When brought to these dispensaries for treatment, many

of the cases are in a moribund condition, yet the mortality is only 2 or 3 per cent.

In the treatment of infantile gastroenteritis with sea-water, there are two important recommendations to be made: first, to avoid all antiseptic internal treatment; and second, to discontinue lavage of the stomach and colon. Should there be constipation, a glycerin suppository is sufficient every second or third day. Sea-water plasma is injected in the scapular region of infants; the gluteal region is so frequently soiled that the scapular region is preferable. The amount injected varies with the weight of the patient. Infants weighing less than six pounds receive 30 Cc. (1 ounce) and those weighing eight to twenty pounds 60 to 90 Cc. (2 or 3 ounces) every two or three days. In grave cases the injections can be given every day, increasing the dose to 100, 180 and 240 Cc. (3 to 8 ounces). For the method of administration, see page 423.

Should the tissues be nearly destitute of water, physiologic saline solution may be administered by hypodermoclysis. A favorable influence on the general symptoms is often produced by the whole pack, with subsequent cool rubbing.

After the bowels have been thoroughly evacuated, the following has been found valuable:

	Gm. or Cc.	
R—Resorcinolis	3 0	gr. xlv
Bismuthi subnitratiss	10 0	ʒiiss
Tannigeni	4 0	ʒi
Misce et ft. pulv. no. xvi.		
Sig.—One every three hours.		

For the relief of pain after the bowels have been thoroughly evacuated:

	Gm. or Cc.	
R—Tinctura opii camphoratae	30 0	ʒi
Bismuthi subnitratiss	15 0	ʒiiss
Mistura cretae q. s. ad	60 0	ʒij
Misce		
Sig.—Teaspoonful every two hours.		

For the relief of pain after the bowels have been thoroughly evacuated, when there is vomiting:

	Gm. or Cc.	
R—Extracti opii	0.06	gr. j
Olei theolomatis, q. s.		
Misce et ft. suppos. no. ii.		
Sig.—Introduce one into the rectum, and repeat in two hours if necessary.		

CHAPTER XXXV.

CHRONIC ENTEROCOLITIS.

CHRONIC INTESTINAL CATARRH—CHRONIC ENTERITIS—CHRONIC COLITIS—CHRONIC SIGMOIDITIS.

ENTEROCOLITIS is a catarrhal inflammation of the mucous membrane of the small intestine and the colon. We should distinguish between primary and secondary chronic intestinal catarrh. One of the most frequent causes of primary intestinal catarrh is an acute enteritis that has not healed or that has been imperfectly treated. Moreover, it is possible for all those injurious agencies which irritate the mucous membrane of the intestine, mentioned as causes of acute catarrh, to give rise to a primary chronic catarrh (errors of diet, frequent colds, habitual employment of laxatives, constipation). Secondary intestinal catarrh generally develops as a consequence of gastric disease, fermentative dyspepsia, or chronic affections of the gut itself (ulcers, tumors, stenoses, and appendicitis).

Pathology. At the beginning of the disease the mucous membrane is swollen and hyperemic. The solitary follicles and Peyer's patches are likewise swollen. In severe cases extravasation of blood may take place. The distended solitary follicles may break and form very small ulcers. The interstitial tissue in such conditions is infiltrated with small round cells. The mucous membrane may become covered with polypoid granulations, and the glands may become dilated and constricted into cysts containing mucus. The glandular epithelium is distinctly loose and swollen so that it is easily shed from its base. When these changes persist for a long time, degenerative changes gradually supervene: many of the glands perish, the mucous membrane becomes attenuated and atrophic, and the interstitial tissue proliferates. Previous hemorrhages are indicated by slaty-blackish discoloration of isolated regions of the mucous membrane. Should the disintegration of the epithelium be very marked, ulcers may form, and if the conditions are favorable they may develop into large catarrhal sores.

Chronic catarrh may affect anatomically all sections of the small and the large intestine. Clinically we distinguish chronic catarrh of the small intestine, chronic catarrh of the large intestine, and combined or mixed catarrh (the small and the large intestine being diseased simultaneously).

Symptoms. The subjective discomforts alone are not sufficient for the diagnosis of chronic intestinal catarrh. These discomforts

vary exceedingly in proportion to the intensity and the extent of the morbid process; sometimes they are entirely absent, and the catarrh is only recognizable by the thin consistency of the stools; on the other hand, there may be more or less distress, though the stools are apparently normal. Frequently the troublesome symptoms disappear completely for a long time. The rule is that the distress, pain, and pressure sensations, at either definite or fluctuating points, are more pronounced and frequent when there is marked diarrhea than under other circumstances, though constipated patients are also subject to attacks of pain, flatulence, and annoying pressure symptoms in the lower bowel. Frequently constipation and diarrhea alternate. The general condition of the body suffers most when there is pronounced diarrhea; in such cases considerable loss of weight may take place. When the catarrh persists, nervous symptoms frequently develop.

Diagnosis.—The diagnosis is determined by the nature of the stool. It must be emphasized that the alvine discharges in catarrh of either the small or the large intestine may be diarrheic or firm, well formed, and on inspection apparently normal. When catarrh of the small intestine is fully developed, diarrhea usually sets in; constipation is rare. In catarrh of the large intestine there is an equal frequency of diarrhea and constipation. The presence of mucus in the feces is characteristic of catarrh of the colon. This mucus is evacuated with the feces or may be demonstrated by a test lavage of the colon. It is always composed of rather large and coarse shreds, and is lighter in color and more transparent in proportion as its origin is farther up the intestine. The higher up in the colon the mucus originates, the smaller are the individual flakes and the more uniformly distributed are they found in the feces. Without the demonstration of mucus it is impossible to diagnose catarrh of the large intestine (see page 121).

In making a diagnosis of chronic intestinal catarrh we should not mistake the brownish mucus which originates in the lowest portion of the rectum, and which is so frequently seen in cases of chronic constipation; the mucus may appear as a thin varnish-like coating upon the hard fecal masses. We must also eliminate the cases where an enormous quantity of mucus is found, as in enteritis membranacea. Catarrh of the lower section of the bowel is probable when there are evacuated hard scybala, embedded, as it were, in membranes of mucus. Furthermore, the test-diet stool in catarrh of the colon, either the diarrheic or the constipated variety, differs from the stool of catarrh of the small intestine, inasmuch as in the former the food is properly digested and it is impossible to demonstrate the presence of biliary coloring matter (see pages 131-132).

The stool is totally different in catarrh of the small intestine. It is generally soft in character and presents evidence, according

to the intensity of the catarrh, of a disturbance in the digestion performed by the small intestine. Schmidt's test diet reveals, on both macroscopic and microscopic examination, defects in the digestion of meat, fat, or carbohydrates, or all three. In these cases, also, the presence of mucus may be often demonstrated. This mucus differs from that produced by the large intestine, however, in that it consists of the most minute, glassy, transparent flocculi, containing few cells, a very few nuclei, and a large number of bacteria. It is frequently discolored by bile, or it becomes green in color on the application of Schmidt's sublimate test, this being absolute proof of origin in the small intestine. Should the sublimate test (see page 116) be negative, bilirubin crystals are occasionally demonstrable microscopically.

The demonstration of bilirubin, *i. e.*, of unaltered biliary coloring matter, by means of the sublimate test, may also be performed on the other constituents of the feces, and a positive reaction always points to catarrh of the small intestine. When the diarrhea is frequent the entire fecal discharge occasionally turns green on the addition of sublimate, or when the passage through the intestinal tract is very rapid it looks green, even without the addition of sublimate, in consequence of its containing unaltered bile coloring matter.

When it is found impossible to prove the presence of mucus on macroscopic examination of the feces, this does not infallibly indicate that there is no catarrh of the small intestine—for it may happen that mucus originating high up in the gut is dissolved and digested before reaching the large intestine (see page 121). Under such circumstances the importance of demonstrating the decomposition of protein in the feces becomes apparent (see page 117). In a stool containing decomposed protein there are found, not so much the decomposed remnants of protein that has escaped digestion, as the albuminous products of the intestinal mucous membrane itself (mucus, pus, serum, blood). In catarrhal processes of the mucous membrane of the small intestine, therefore, mucus and serum, even when they cannot be demonstrated macroscopically, may be shown with certainty to be present by proving the existence of decomposed protein in the feces. This proof is afforded by the incubator test (see page 116), in which an alkaline reaction is developed, with darkening of the feces and generation of gases. The same conclusion is arrived at when protein is found in the filtrate of the feces by the acetic-acid-boiling test and the potassium ferrocyanid test. Since, according to the preceding statements, putrefaction takes place in the majority of cases of catarrh of the small intestine, it follows that the diarrhetic stool in such cases is nearly always of a dark brown color, of alkaline reaction, and of the most disagreeable, putrid odor. Actual fermentative stools of light yellow, frothy quality may be passed when the digestion of carbohydrates is

markedly insufficient and the production of mucus and serum is insignificant. These cases, however, are rather rare. But even in these cases an exact analysis of the feces will reveal the signs of catarrh. Feces that look normal may be passed after test meals in cases of slight catarrh of the small intestine; but an exact analysis will demonstrate the presence of mucus or decomposed protein. In mixed catarrhs these two varieties of stools may be combined. Diarrheas in such cases are quite frequent. An exact fecal analysis is therefore essential for diagnostic purposes (Plate VIII).

Blood and pus are found in the feces in ulcerations and hemorrhages. Blood shows itself either macroscopically, as bloody mucous flocculi and pure blood, or chemically, when the diet is meat-free, by the benzidin test. Pus appears in the form of markedly purulent mucus or in small yellowish-white globules, which are plainly visible when the triturated fecal matter is placed on a black plate (Plate VI). The pneumatic sigmoidoscope is often helpful in the diagnosis (Fig. 46).

Prognosis.—The course of chronic enteritis is always long and tedious. There can be no doubt that recovery does take place when proper treatment is faithfully persevered in. Many cases, however, can only be improved, and relapses may occur from indiscretion in diet. When severe catarrh and diarrhea persist, the general condition may become greatly disturbed, resulting in decided loss of weight. Complication with ulcers occasionally causes severe symptoms, and will be considered more fully under its proper heading.

Treatment.—*Treatment of the Cases with Diarrhea.*—As in other intestinal diseases, the principle of giving rest to the affected organ must govern our therapeutic measures. It is very important that patients with pronounced catarrh should be placed in bed for several weeks, the mind also being kept as quiescent as possible. Only in light cases can bodily and mental exercise be permitted. In the moderately severe and especially in grave cases a regular "cure" should be insisted upon if the circumstances of the patient will at all permit. Such a cure can best be undertaken in a private hospital in which the medical direction is reliable and the required therapeutic measures can be properly applied. The results of treatment with patients who are compelled to follow their usual occupations are, naturally, far less satisfactory. In such cases, which unfortunately are always in the majority, as much bodily and mental rest as possible should be insisted upon. All kinds of sport, swimming, rowing, bicycle riding, horseback riding, golf, etc., must be interdicted.

A carefully selected, non-irritating diet is of the greatest importance; for complete details see page 172. When the small intestine is affected, the diet must be guided by the results of examina-

tion of the feces after a test diet; it should always be antiputrid, because in most cases of catarrh of the small intestine putrefactive processes are well marked. Antifermentative diet will also have to be considered, though not so frequently. It is not necessary to be quite so careful with the diet in cases of diarrhea with catarrh of the large intestine. This is especially true when it is known that the small intestine is healthy, for in such cases even solid foods are sufficiently broken up before reaching the colon. The small intestine must always be considered in the treatment of cases of mixed catarrh.

Zweig gives the following diet:

EARLY:	Acorn cocoa in water; saccharin, toast, butter.
FORENOON.	One or two eggs, toast, butter.
NOON:	Soup, such as rice, oatmeal without salt, minced meat or fish boiled in butter; whortleberry jelly, rice or hominy; noodles or macaroni, one or two glasses of whortleberry wine or claret, toast.
AFTERNOON:	As in the forenoon.
SUPPER.	Soup as at noon; minced meat, whortleberry jelly, one glass of whortleberry wine or claret, toast, butter.

Boss prescribes for cases of moderate severity the following diet:

8 A.M.	Acorn cocoa in water; saccharin, toast and butter (20 to 30 Gm.).
10 A.M.	One cup (200 Gm.) of porridge of rice or oatmeal, wheat meal in veal bouillon (without salt), 50 Gm. roast veal or beef (minced), or fried fish or cold meat (no ham).
1 P.M.	Thick soup, legume, oatmeal, hominy with the addition of nutrose, tropon or eucasein; whortleberry soup, 200 Gm.; bouillon, rice or hominy (cooked to semisolid consistency), vegetables or potatoes mashed, 50 to 100 Gm.; meat, fish (50 to 100 Gm.), butter gravy. Pudding containing a little yolk of egg and saccharin. (All fruits except whortleberry jelly are forbidden. No fruit juices.) Beverages—whortleberry wine, Burgundy, Camarate, Lima Rula wine and old claret. (Sweet wines, champagne and effervescent drinks are not permissible.)
4 P.M.	Tea, cocoa, biscuits, toast, zwieback with butter.
7 P.M.	Thick soup. Cold or warm meat, 50 Gm.; toast, butter (20 Gm.). One or two glasses of whortleberry wine or claret.
9 P.M.	One glass of hot whortleberry lemonade or a hot punch with saccharin, or tea with claret.

When the dietary measures are not adequate, medication may be necessary. The calcium preparations are especially recommended—calcium carbonate and calcium phosphate, of each equal parts, one teaspoonful three times daily in water. This may also be given in combination with bismuth subnitrate.

	Gm. or Cc.	
R—Calcii carbonatis,		
Calcii phosphatis	33 30 0	3j
Bismuthi subnitratiss	15 0	3ss

Misce.

Sig.—A teaspoonful three times daily, stirred in water.

	Gm. or Cc.	
R—Calcii carbonatis	30 0	3j
Bismuthi subgallatis	4 0	5j

Misce.

Sig.—One teaspoonful three times a day, stirred in water.

If the intestinal catarrh is complicated with hyperacidity, the calcium preparations are better than the sodium preparations, because the latter may increase the diarrhea. The treatment with intestinal sedatives is fully described on pages 274-281. In the presence of fermentation the following combination may be used:

	Gm. or Cc.	
R—Resoremolis	4 0	3j
Bismuthi salicylatus	6,0	3 iiss
Misce et ft. chart. no. xii.		
Sig.—One before each meal.		

It must not be forgotten that chronic enteritis may be caused by microorganisms or their toxins. The colon bacillus is a frequent offender. An autogenous vaccine would be valuable if one could be prepared. A polyvalent stock bacterial vaccine containing 50,000,000 colon bacilli to the dose, or the "sensitized" vaccine, can be given subcutaneously once a week with absolute safety. The interval between doses may be reduced and the dose increased if little or no reaction, local or general, follows the last preceding dose.

Kaolin (bolus alba) has given good results in a dose of 60 to 100 Gm. (3ij-ijj). It should be taken in a large volume of water in the morning, before breakfast.

In abdominal pains, opium is inadvisable and should only be administered for the relief of an irritating tenesmus; the suppository is the preferable form. Lavage of the rectum is advantageous in some cases of catarrh of the intestine, and should be given as described on pages 232-236. Enemata of Carlsbad water or whortleberry decoctions, or of insoluble astringents such as bismuth, are frequently of great benefit. Enemata containing tannic acid or nitrate of silver are inadvisable.

The injection of hot gelatin solution will frequently bring about a complete cure of severe chronic catarrh of the large intestine, with diarrhea, which has resisted all other measures. The sphincter ani and a short distance above are extremely sensitive to heat, and consequently the solution is injected through a tube about four inches long, with the patient on his left side. From 30 to 60 Cc. of a 10-per-cent. solution of gelatin in water, at a temperature of 113° to 125° F., is injected. The patient lies still for two hours afterward, with heat applied to the abdomen. Gelatin has a soothing action on the inflamed mucous membrane, while the heat induces a healing hyperemia; the calcium present has also a favorable influence on the local repair. It is possible that the gelatin may check the secretion of readily putrefying material from the intestinal walls. This assumption is sustained by the prompt arrest of the diarrhea and rapid transformation of a strongly positive into a negative response to the incubator test (see page 116). The daily injections should be continued for eighteen to twenty-eight days.

The use of mineral waters is frequently indicated and quite

cious. As already stated, Carlsbad water has a curative effect in chronic catarrh (see page 264). If Carlsbad water is to be used for any length of time, it should be taken in small quantities only, twice daily, or in much smaller quantities, 30 to 60 Cc. (3j-ij), three times a day, very hot. The cure taken once, for four weeks only, is not enough; it should be taken at least four times during the year, one month at a time, at home. The waters of Saratoga have given good results in cases of chronic catarrh of the intestine. The drinking of the calcium waters has also proved quite effective. Ferruginous waters are to be employed in anemic conditions (see page 254).

While the patient is resting in bed, warm applications (wet or dry), thermophores, or Priessnitz bandages are to be applied to the abdomen during the day. The abdomen must be kept warm. It is also very advisable that a warm abdominal bandage be worn for some little time after this resting period is over. Rubbing, washing, sitz baths and packs are to be used in addition. (See Chapter XII.)

Treatment of the Cases Associated with Constipation.—Here, also, both bodily and mental rest are quite essential. It is not necessary, however, to insist on absolute rest in bed in moderate or light cases. The diet should be calculated to remedy the constipation, as outlined in Chapter VII (see pages 182-186). In this pathologic condition, however, the diet must act only chemically on the constipation; mechanical irritation by a coarse diet rich in cellulose must be entirely avoided. The cellulose remnants may be adequately replaced by large quantities of agar, 30 to 60 Gm. (1 or 2 ounces) per day, which render the stools soft and mushy and do not irritate the mucous membrane. As constipation is found only in catarrh of the colon, the diet may be a little heavier than if the small intestine were involved. It is usually possible to regulate the stool by means of the diet, and thereby to diminish the production of mucus in the colon.

Should dietetic measures fail, enemata should be used as adjuvants. A solution containing table salt, lime-water (three or four tablespoonfuls to a liter of water) or bicarbonate of sodium is useful.

Purgatives are strictly to be avoided. Liquid petrolatum (refined mineral oil) deserves consideration in such cases—its softening effect on the fecal matter being very desirable, as is also its emollient effect upon the mucous membrane. The daily dosage is two or three tablespoonfuls. This oil (see page 664) has been found to be particularly beneficial. The following prescriptions have been found useful:

In hyperacidity with diarrhea:

	Gm. or Co.	
R—Bismuthi subnitrat̄is	15 0	3ss
Calcii carbonatus precipitat̄is		
Cretæ preparatæ	35 30 0	3j
Misce.		
Sig.—Teaspoonful in water one hour after meals.		

In hyperacidity with constipation:

	Gm. or Co	
R—Sodii bicarbonatis	45 0	3iss
Magnesi oxidi	15 0	3ss
Misce		
Sig. —Teaspoonful in water one hour after meals		

For abdominal pain:

	Gm. or Co	
R—Anesthæsi	4 0	3j
Misce et ft caps no xvi		
Sig. —One every three hours.		

For tenesmus:

	Gm. or Co.	
R—Extracti opii	0 06	gr. j
Extracti belladonnæ	0 03	gr. ss
Olei theobromatis, q. s.		
Misce et ft suppos no iii		
Sig. —One introduced into the rectum every hour if necessary.		

As an astringent and intestinal antiseptic:

	Gm. or Co	
R—Tannopini	15 0	3ss
Misce et ft pulv no. xvi.		
Sig. —One every three hours.		

Ionization with a 2-per-cent. zinc sulphate solution has been found efficacious in the treatment of chronic enterocolitis with or without ulceration. The method of application is simple, easy, and painless. The bowels should be washed out with warm water, to free them as much as possible from gas, fecal material, blood and mucus. A large pad, 8 by 10 inches, covered with cloth, is dampened with warm saline solution, then attached to the negative pole of a battery and applied to the abdomen. A rectal electrode (Fig. 38) is lubricated and inserted into the bowel just above the sphincters. A rubber tube attached to an ordinary irrigator is slipped over the free end of the rectal electrode. The irrigator is fed with a warm 2-per-cent. zinc solution. The solution is allowed to flow into the bowel until it is estimated that the whole of the diseased part is filled with it. The positive wire from the battery is then fixed to the binding screw of the rectal electrode, and the current is turned on slowly up to 15 to 30 milliamperes, according to the tolerance of the patient, and allowed to act for ten to fifteen minutes. If the bowel is irritable it is advisable to first insert an opium suppository. A bed-pan should also be placed under the patient in case there should be an urgent call to empty the bowel. This treatment can be carried out at first daily, then every other day, and later twice a week. The mucus and blood will soon disappear from the stool, and the colonic pain will be greatly alleviated. After the treatment is finished and the rectal electrode is removed, the zinc solution is evacuated by the bowel in the ordinary way. (See Chapter XI.)

CHAPTER XXXVI.

ENTERITIS MEMBRANACEA.

MUCOMEMBRANOUS ENTERITIS—MUCOUS COLITIS—PSEUDOMEMBRANOUS ENTERITIS—TUBULAR DIARRHEA.

THE morbid process known as enteritis membranacea owes its name to the peculiar spastic evacuation of considerable quantities of mucus which characterizes it. There are two theories concerning the etiology of the disease: first, that the spasmodic excretion of mucus is a secretory neurosis (colica mucosa, colica mucomembranacea, myxoneurosis intestinalis mucomembranacea) without anatomic affection of the intestinal mucous membrane (a condition comparable to bronchial asthma); second, that the cause of the disease is catarrh of the colon (colitis membranacea, enterocolitis mucomembranacea). Recently a compromise view has gained acceptance, it being assumed that in enteritis membranacea there are both nervous irritation and membranous inflammation. The inflammation may be circumscribed, confined to small regions of the mucous membrane of the colon, and giving little evidence of its existence. There is always a derangement of the vegetative nervous system (see page 387). These patients respond to all the tests for vagotonia. There is an overstimulation of the vagus, probably through the internal secretions. The nervous irritation may emanate from the genitals, from mental influences, from constipation, or from coloptosis. Hypersensibility toward certain foods or certain changes in temperature, or toward particular positions or movements of the body, may produce a reflex nervous irritation. It is evident that persons of nervous temperament are particularly predisposed to this malady. Enteritis membranacea, however, does undoubtedly occur in persons who are by no means "nervous;" it cannot be viewed as a local symptom of either neurasthenia or hysteria. This is proved from the fact that the disease is occasionally induced by irrigating the intestine with irritating astringents such as silver nitrate or tannic acid.

Pathology.—Up to the present time no pathologic-anatomic basis has been discovered for this disease. All authors agree, however, in their description of the characteristic symptoms of the disease—the seizure-like evacuation of mucus, the disturbances of fecal evacuation, and the painful intestinal contractions. Women constitute from 80 to 90 per cent. of all cases; the age incidence is usually between twenty and forty. The patients are generally

anemic, weak, exhausted by much child-bearing. The affection is rare in advanced age and very rare in childhood, so far as the published reports show.

Symptoms.—Constipation is a feature of the large majority of cases, and diarrhea is rare. Defecation is usually attended with pain and the expulsion of mucus. The pains come on beforehand, and may assume an exceedingly violent, colicky character; relief is afforded by the evacuation. The feces often resemble those of sheep, and suggest spastic constipation (see page 668). Simultaneously with the fecal evacuation, large quantities of mucus in the shape of the well-known bands are expelled. This mucus may be ribbon-like, three or four inches long, in irregular shreds of various shapes, or in the form of regular hyaline casts of the intestinal tube. Occasionally the evacuation of mucus takes place by itself—the passage contains nothing but mucus. The course of events is by no means the same in all cases. Occasionally the pains are very slight or even entirely absent, and sometimes they are not experienced with every act of defecation. The expulsion of membranes may alternate with that of fecal matter, resulting in most heterogeneous aspects. Other objective signs may be present, such as occasional tenderness to pressure over the large intestine; this, however, is not characteristic of the disease. After the disease has continued for some time the patients usually have pronounced general nervous symptoms, which, coming on in this manner, must be regarded as a consequence of the affection (see page 133).

The course of the disease is distinctly chronic. The trouble often lasts for many years, although frequently it is interrupted by periods of from one month to three months of well-being. With proper treatment cures are quite likely to be accomplished.

Treatment.—The treatment is directed against the fundamental disease, if such be recognized with certainty or even with probability. Therefore great stress must be laid on the proper general treatment of nervous patients by means of hydrotherapeutic, physical and electrical measures. Patients deficiently nourished are to be subjected to an invigorating regimen; hyperalimentation (see page 569) is especially beneficial in such cases. Should enteroptosis be present, it is to be treated by suitable abdominal bandages (see page 574); and anemic conditions are to be counteracted by the usual means. Mental irritation must be avoided as much as possible.

This disease is closely connected with nervous dyspepsia and with enteroptosis. The intestinal condition is directly dependent on the failure of the stomach to perform its functions, and therefore therapeutic measures should be directed to the basic affection. The abnormal condition of the stomach (vagotonia) irritates the solar plexus, and this irritation is transmitted to the intestinal plexus and thus causes the intestinal symptoms. The anom-

of the gastric functions (principally achylia) and enteroptosis exist in a large number of cases of membranous enteritis. As movable kidney and enteroptosis are so frequently found in cases of membranous enteritis, the correction of these displacements must not be neglected. (See Chapter XXX.)

In cases associated with anemia the intramuscular injection of citrate of iron, 0.05 Gm. (1 grain), is a measure of great advantage. The hemoglobin will increase more rapidly under this method of treatment than under any other. Arsenic and the glycerophosphate of sodium can be combined with the citrate of iron when indicated. The cacodylate of sodium or iron, 0.05 Gm. (1 grain) intravenously, is an alterative and acts well on the metabolic processes (see page 581).

These measures are intended to combat the nervous irritation which is so closely related to the characteristic symptoms. The inflammatory part of the disease, viz., the more or less pronounced circumscribed cellulitis, and the constipation, require careful consideration. The regulation of the diet is of prime importance, and with this in view various suggestions have been offered. It is evident that the diet will have to be abundant, in order to increase nutrition as much as possible and antagonize the constipation. A non-irritating diet is by no means indicated, for the small intestine is normal and is able to cope with an abundance of hardy food. Some authors, *e. g.* von Noorden, propose, as applicable throughout the entire course of enteritis membranacea with constipation, a diet consisting of coarse articles rich in cellulose (coarse bread, legumes, fruit containing small seeds and thick skins, vegetables rich in cellulose), as described in Chapter VII on Diet. Fat should always be a prominent part of the diet. The commencement of this dietetic treatment should be quite abrupt. Zweig publishes a list which corresponds essentially to this kind of diet:

EARLY	Tea with milk Graham bread, butter, honey
FORENOON:	One glass of one-day kefir Pumpernickel, black bread, butter.
NOON:	No soup Meat or fish; vegetables, salad with vinegar and oil, pudding with sweet fruit juice, jam, fruit (grapes, dates, figs, oranges), Graham bread Apple juice, one glass
AFTERNOON:	Chocolate with whipped cream Graham bread, butter, marmalade
EVENING.	Eggs or bacon and eggs, meat (cold or warm), salad; jam; Graham bread, butter, cheese, fruit. Apple juice, or one glass of white wine
10 P.M.	One glass of kefir (one-day).

The coarseness of this diet, however, might in some cases lead to irritation of some portion of the mucous membrane of the colon which is already irritated. Many authors therefore advise a constipating diet, more stress being laid upon its chemical action than upon its richness in insoluble remnants of food. The type of this diet is the one given in cases of spastic constipation (see page 185), in which the percentage of calories is increased as much as

possible by the addition of fat. In case it is desired to add more insoluble ingredients, the procedure of Schmidt might be followed—the daily administration of a considerable quantity, 30 to 60 Gm. (1 or 2 ounces), of finely divided dry agar; an alternative method would be to give the agar in the form of jelly-like foods. This material renders the stools soft, mushy, and rich in water. It represents a kind of coarse food, the irritation caused by it being slight, however, in comparison with that caused by hard cellulose remnants; it is well adapted to assist the purgative action of a chemically active diet. The addition of gelatin to the food might likewise be indicated. Quite recently it has become customary to recommend a diet poor in meat, or, better yet, one free from meat, *i. e.* a lactovegetable diet (see page 422). The results obtained from this diet have been quite good.

Ewald prescribes the following lactovegetable diet:

EARLY:	$\frac{1}{2}$ liter sweet cream or cocoa or oatmeal-cocoa; white or black bread with butter; honey, marmalade, or fresh fruit.
FORENOON:	Porridge of rice; lentils, hominy; milk, kefir; white or black bread with butter.
NOON:	Leguminous or fruit soups (apple, plum, cranberry, raspberry, gooseberry, cherry); vegetable soups (spinach, carrot, tomato); milk soups or cold soups; fruit juices; gooseberries; black bread and raisins (small), buttermilk or sour milk. This is to be followed later by plenty of green vegetables (with as much butter as agreeable), or instead of the vegetables mashed peas, lentils, rice with apples if desired), dried fruit with dumplings, macaroni, pudding melange with fruit sauce, salads, egg dishes, bread and butter, fresh cream cheese.
AFTERNOON:	Boiled or fresh fruit plentifully, with zwieback or white bread; honey or fruit jellies.
EVENING:	Thick soup of barley, oats, rice, hominy, tapioca, etc.; fried potatoes; butter, cheese, eggs and dishes prepared from eggs, milk, etc.

Should diarrhea be present, a residue-free diet is indicated, embracing especially cream, zwieback, rice, and chocolate. Coffee, wine and tobacco are to be forbidden.

The enteritis must receive attention, and be properly treated locally apart from dietetic measures. The painful crises can frequently be relieved by the use of pancreatin in combination with bile. Sodium bicarbonate can be added. Not only the pain but the character of the stools seems to be relieved by this combination.

Fleiner's oil enemata, used constantly, have given good results (see page 226).

Irrigations of the large intestine with 0.9-per-cent. sodium chloride solution are likewise recommended, and especially irrigations with sodium bicarbonate, Carlsbad water, and solutions of the Carlsbad salts. Astringents, especially silver nitrate and tannin, are strictly contra-indicated. Duodenal lavage is especially advantageous (see page 105).

The main clinical feature of membranous enteritis is great irritability of the muscular coat of the intestine. This may result either from undue irritation of the autonomic nervous mechanism or from local causes. As to the local irritant, this may be either some toxin absorbed from the bowel or some substance circulating in the blood. The colon contains an abnormal amount of some irritating toxin or other substance which normally is present in small amounts or not at all; the excess is due to bacterial activity.

An autogenous bacterial vaccine should be of great benefit in this condition. Good results are often obtained from colon bacillus vaccine. The pain and mucus diminish, and the general health improves. The colon is particularly hospitable to bacteria, containing, even under normal conditions, a much greater number than any other part of the intestinal tract. One-third of the weight of dry feces is said to consist of bacterial bodies.

When it is impossible to regulate the bowel evacuation by means of dietetic measures alone, it will be necessary to occasionally resort to laxatives. Only mild laxatives are to be employed, however, *e. g.*, castor oil, rhubarb, tamarind, and liquid petrolatum. Cascara-agar is even better than any of these, because it exerts an agreeable and to a certain extent peculiar non-irritating, softening and exceedingly mild laxative action (see Chronic Constipation, page 659). A considerably smaller quantity of cascara-agar is required than of pure agar, one or two tablespoonfuls per day being sufficient. Atropin is a most efficacious therapeutic agent in enteritis membranacea, a manifestation of vagotonia (see page 388). In case of intestinal spasms with subsequent constipation and pains, narcotics may be necessary and may be used, as opium and belladonna suppositories. The extract of belladonna, 0.0075 to 0.015 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain) three times a day, is very valuable. Hot carminative teas have a modifying effect on the spasm. The bromids are, as a rule, of little value. Papaverin and benzyl benzoate are valuable antispasmodics (see page 276).

Hydrotherapeutic measures are useful adjuncts in the relief of attacks of pain, *e. g.* warm abdominal packs and warm sitz baths (100° F.). Half-baths, douches, water pourings, packs, climatic changes, or sojourn in the mountains or at the seashore may be of great benefit. Extended vacations far away from business cares are always favorable to recovery (see page 247).

Very light massage of the abdomen (effleurage) is occasionally useful, as is also the percutaneous application of a weak galvanic current (see page 211).

High-frequency currents are beneficial in some cases of mucous colitis. High-frequency treatment is given on the condensor couch, through the hands, for ten minutes, followed by fifteen minutes' local application, sometimes from the low tension and sometimes from the resonator. This results in improvement of the

General condition, and the pain gradually diminishes. The treatment should be continued for several months to accomplish a complete cure.

Sea-water plasma injections have given good results in some cases of enteritis membranacea. The beneficial effect is brought about through the medium of the nervous system. After five or six injections pain and the excretion of mucus cease and the evacuations become well formed and regular. The distention and weight in the abdomen gradually subside without the necessity of a rigid diet.

The injections are first given in doses of 30 Cc. (3j) every two or three days. Should the patient be extremely nervous, it is better to begin with 20 Cc. Should there be any temperature or general malaise after the injection, the dose should be diminished or the interval between the injections lengthened. In case there is no reaction and no improvement in the digestive symptoms, the amount of sea-water administered at a single injection is to be gradually increased to 40, 50, 75, and 100 Cc. These injections should be given intramuscularly in the gluteal region, preferably at bedtime. The treatment can be continued during the menstrual period (see page 423).

Mineral-water cures are not indicated; they overstimulate secretion and often do a great deal of harm.

The treatment of enteritis membranacea will always be most successful when it is possible to remove the patient from his domestic surroundings and to place him in a private institution that is devoted especially to the treatment of this class of cases, and where all the therapeutic apparatus is at hand. This, unfortunately, is impossible in general practice, as the greater number of the patients are compelled to be actively engaged either at home or in business; furthermore, these "cures" in special institutions always demand considerable pecuniary expenditure.

Attempts have been made to cure the disease by surgical means, such as the establishment of a temporary artificial anus, temporary colotomy, or partial resection of the colon. The results obtained by these procedures are, however, of a very doubtful nature.

The clinical evidence in favor of surgical intervention in cases of mucus colitis is not convincing. Cases of chronic constipation with colonic irritation are at times diagnosed as true membranous colitis, and have been cured by colonic irrigation. Some surgeons advocate colonic irrigation through appendicostomy (Fig. 104). The vermiform appendix is fixed to the abdominal wall and its lumen used to introduce fluids into the cecum for irrigating the colon. The irrigating fluid passes through the whole of the large intestine and is evacuated by way of the rectum.

A chronic appendicitis may occasionally be the cause of mucous colitis. There is no doubt that the removal of a chronically inflamed appendix will assist in the recovery of some patients.

The following prescriptions have been found useful:
To relieve pain due to intestinal spasm:

	Gm. or Co.	
R—Pulveris opii	0 03	gr. $\frac{1}{2}$
Extracti belladonnæ	0 008	gr. $\frac{1}{4}$
Extracti hyoscyami	0 015	gr. $\frac{1}{4}$
Misce et ft. suppos. no. i.		
Sig.—To be introduced into the rectum.		

For the relief of vagotonia:

	Gm. or Co.	
R—Extracti belladonnæ	0 125	gr. ij
Misce et ft. caps. no. xvi.		
Sig.—One three times a day.		

Carminative tea to relieve enterospasm:

	Gm. or Co.	
R—Herbæ matricariæ	30 0	5j
Seminis feniculi	4 0	5j
Herbæ thymi	30 0	5j
Herbæ melissæ	30 0	5j
Misce.		
Sig.—Dessertspoonful in a cup of boiling water as a tea.		

Mild laxative:

	Gm. or Co.	
R—Extracti belladonnæ	0 125	gr. ij
Phenolphthaleini	1 0	gr. xv
Pulveris aromatici	2 0	gr. xxx
Misce et ft. caps. no. xvi.		
Sig.—One or two capsules at night.		

For local application during painful colics:

	Gm. or Co.	
R—Olei hyoscyami compositi	100 0	5iij
Extracti opii	5 0	5j
Extracti belladonnæ	1 0	gr. xv
Chloroformi	20 0	5v
Chloralis hydratis, Camphoræ, Mentholis	5â 1 0	gr. xv
Misce.		
Sig.—Saturate flannel and apply to abdomen.		

CHAPTER XXXVII.

CHRONIC CONSTIPATION.

ATONIC CONSTIPATION; SPASTIC CONSTIPATION; FRAGMENTARY CONSTIPATION.

CHRONIC constipation consists of infrequent or difficult evacuation of the bowels. It must not be confounded with obstipation, which is usually from a mechanical cause, such as strictures, angulations, adhesions and tumors. (See Chapters XLVI, XLVII and XLVIII.) Chronic constipation may be divided into three varieties:

- I. Atonic Constipation.
- II. Spastic Constipation.
- III. Fragmentary Constipation.

ATONIC CONSTIPATION.

Primary atonic constipation is one of the most common diseases met with in general practice. It is not peculiar to either sex, though found more frequently in females than in males. The etiology is apparently not the same in every case. Nothnagel held the cause of primary atonic constipation to be an abnormal innervation of the intestine—an abnormal tonus of the peristalsis of colon and rectum, followed by atony. He also asserts the occasional occurrence of insufficient muscular activity of the intestine in consequence of hereditary atrophy of the intestinal muscle fibers. Emminghaus has found degenerative changes in the splanchnic nerves. There can be no doubt, however, that alimentary errors may cause chronic constipation; for example, an improper meat diet, an insufficient quantity of food, and irregularities in the time of eating. Chronic constipation may be a part symptom of a disturbance of the vegetative nervous system (see page 387), as vagotonia or sympathicotonia, of neurasthenia or hysteria, or of the enteroptotic habit. The latest theory, which explains a large number of cases of chronic constipation, has been propounded by Adolf Schmidt, Strasburger, and Lohrich. Schmidt and Strasburger had noted that the test-diet stool of atonic constipation patients contained a remarkably small residue of food, much less than the test-diet normal stool, on both macroscopic and microscopic examination. Lohrich, after having made a systematic study of absorption, explains this peculiar phenomenon as follows:

When the feces of individuals suffering from chronic constipation and those with normal evacuation, both having been fed identically, are collected within the same space of time, it is found that both the moist and the dried quantities of fecal matter are only half as large in those suffering from constipation as in the normal individuals. This small quantity of fecal matter found in chronic constipation depends on the fact, as is shown by analysis, that the feces of constipation contain only half as much protein, fat, carbohydrate and cellulose as the normal feces under a similar diet. The small amount of moisture is due not only to a larger loss of water from prolonged retention of the feces in the bowel, but also to an extraordinarily effective utilization of the food. This utilization, compared with that in health, is too effective. Simultaneously there are absent from such constipation feces all the phenomena of putrefaction and of fermentation. One effect of this too marked utilization of the food is that the intestinal bacteria of fermentation and putrefaction cannot flourish in so poor a nutritive medium as the exhausted feces, and consequently the intestine is deprived of the stimulus of large quantities of fermenting putrefactive products, with the result that intestinal motility is retarded and atonic constipation follows. The correctness of these researches has been confirmed by other authorities, and thus an explanation is afforded for a very large proportion of those cases of chronic constipation in which small quantities of hard fecal masses, dry and modulated, are evacuated, at long intervals—eight to fourteen days in obstinate cases. This theory, of course, does not apply to all forms of atonic constipation, particularly those cases in which the feces remain soft and marked processes of decomposition (flatulence) take place, for in these cases muscular relaxation of the rectum and large intestine must be assumed.

Symptoms. The symptoms of chronic constipation are well known. The most characteristic and prominent symptom is irregularity in the evacuation of the bowel, this act taking place not daily, but at intervals of two, three or even more days, and then only with the aid of strong abdominal pressure on the part of the patient. The patients sometimes feel perfectly well, notwithstanding all this; often, however, they experience a variety of discomforts, especially of a nervous nature. They complain of headache, pressure in the head, congestion of the head, loss of appetite, mental depression, and hypochondriasis—all of which are improved when the bowels have been evacuated (see page 419).

Disturbances of this kind are frequently associated with a very limited ingestion of food—a fact which increases the evils, especially when the emaciation is accompanied by ptosis of the abdominal organs. In this manner a vicious circle may become established, the origin of which it is impossible to trace. Colicky pains are rare in atonic constipation. Light, moderate and severe cases are

to be differentiated according to the length of the intervals between the fecal evacuations, the degree of the subjective symptoms, and the manner in which the bowel reacts to therapeutic measures. In very obstinate and neglected cases fecal impactions may form in the cecum and in the hepatic and splenic flexures. The hard fecal matter may become firmly lodged in the recesses and in the ampulla recti (dyschezia), so that softer fecal matter passes by without dislocating it (Plate XX, Fig. 4). These fecal impactions occasionally give rise to symptoms of stenosis and ileus.

The test-diet stool of chronic constipation with perfectly dry feces is very small in volume, consisting of various sized hard nodules and lumps which are partially coated with a thin, varnish-like, shiny layer of rectal mucus. Macroscopically, no food remnants of any kind are recognized, not even remains of cellulose. Microscopically, as compared with normal fecal matter, remarkably few food remnants, such as small muscle fragments, potato cells, fat, and minute remains of cellulose, are visible. The fecal matter is of neutral reaction and odorless; in the incubator no gases are formed, and even there its reaction remains unaffected. In cases in which there is a muscular relaxation of the intestine and the feces remain soft, the test-diet stool corresponds to the test-diet normal feces (see page 133).

Besides primary atonic constipation there are a number of secondary varieties which may supervene as a result of gastrointestinal morbid conditions or diseases of other organs. The test-diet stool affords diagnostic data when the constipation is due to catarrh of the intestine.

Roentgenograms after a bismuth meal or after a large enema of bismuth emulsion will sometimes give valuable information (see page 146). We must not depend too much on these alone. They are of value only as confirmatory evidence in constipation. The Roentgen fluoroscope is more helpful, but in obstipation rather than in constipation.

Treatment.—Parents and educators should endeavor to supervise the bowel functions of childhood; to insist that the stools be punctually evacuated by accustoming the child to a fixed hour daily; and, in case of necessity, to establish regularity in defecation by adequate dietetic measures. Purgatives are not permissible in childhood. Constipation is apt to accompany pregnancy, long rest in bed, infectious diseases, and convalescence after surgical operations. In all these conditions it is essential to carefully watch the action of the bowels, and to regulate them as occasion arises.

The aim of the treatment must be to so improve the constipated condition by dietetic measures as to finally attain regularity of defecation with a normal food supply. The diet in atonic constipation is fully explained in Chapter VII (see page 182), in respect to both its nutrient composition and its aims. The assertion that it

should be rich in residue has in view the desirability of making the feces as voluminous and rich in water as possible. The addition of plenty of buttermilk, one-day kefir, or yoghurt increases the purgative effect. When there are present simultaneously gastric disturbances, intestinal catarrh, or other diseases (*c. g.*, diabetes), the diet should be adapted to such abnormal conditions, always bearing in mind the desirability of as great a purgative effect as possible.

Blunzel and Ulrici employ a wheat bread containing fine sawdust. The finely sifted sawdust of beechwood is recommended in the proportion of one ounce to one pound of dough. The bread may have as much as 10 per cent. of sawdust without differing in taste or appearance from ordinary bread. Good results were obtained by the authors in 80 cases, it being found that, although the added cellulose was not digested, it increased the peristaltic movements and the amount of the fecal mass.

Bran is frequently used with good results in chronic atonic constipation. The proportion of tough cellulose, dense woody fiber, in bran is said to be 20 per cent. On account of this cellulose residue, the bran directly stimulates peristalsis and forms sufficient bulk for the contracting intestine to push forward. The laxative property of Graham bread, whole wheat bread, and rye bread depends upon the cellulose they contain.

The new theory of Schmidt, Strasburger, and Lohrlich, mentioned above, has brought about a new treatment of atonic constipation. The researches of Lohrlich established the therapeutic indication of rendering the feces more voluminous, softer and more liquid, thus bringing into play the physiologic stimulation of peristalsis. A wisely chosen dietary stimulates peristalsis both chemically and mechanically. Often, however, the dietary prescriptions are not effective, either because the intestine is too much weakened or because the patients do not partake of the diet in the prescribed manner. It was therefore desirable that some agent should be discovered which would fulfil the above-mentioned indications, that is to say, a substance voluminous, rich in water, and hard to digest, and which, if possible, would retard the absorption of the other constituents of the food, especially those containing cellulose. This substance has been found by Schmidt in agar, as explained in Chapter XIV on Medication. Agar absorbs and retains moisture, thus permanently increasing its own volume.

Experiments made in the laboratory of Lafayette B. Mendel, of Yale University, have verified the findings of Schmidt. While ordinary carbohydrates are utilized perfectly in the alimentary tract of man, a considerable number of carbohydrates, such as are contained in certain seaweeds, are not attacked by the digestive enzymes. Experimenting with agar, Mendel found that the greater part was excreted in the feces unchanged. As agar absorbs water

readily and retains it, and as it is able to resist the action of intestinal bacteria as well as the enzymes, its value in the treatment of chronic constipation is apparent. It gives bulk to the stools without introducing any objectionable products of decomposition.

Agar may be employed as an adjunct to a diet rich in refuse. It is usually administered dry and minutely cut up. In order to obtain good results it is essential to take the agar in large doses—30 to 40 Gm. (1 to 1½ ounces) every day, regularly. This quantity is so large in volume as to almost preclude the possibility of its being ingested, dry agar being very light in weight; but even if so large a quantity were regularly taken, the purely mechanical property of the agar would not always be sufficient to bring about a movement of the bowels, since agar alone does not augment the processes of putrefaction and fermentation in the intestine. Schmidt has removed this objection in a very effective manner by the introduction of cascara-agar, sold under the trade name "regulin." Cascara-agar consists of finely cut-up agar with the addition of a small quantity (about 2.5 per cent.) of an aqueous fluidextract of cascara sagrada from which the bitter principles have been removed. The cascara is present in so small a quantity that it has no purgative action; it is liberated in the intestine, where it simulates the stimulating effects of the natural decomposition products of the intestinal contents which are lacking in atony of the bowel.

Agar is not a purgative, but is to be viewed as a means of increasing the efficacy of a laxative diet rich in refuse. It does not move the bowels promptly, but must be taken regularly, daily, for a long time and in sufficient doses. Agar belongs to the diet in constipation just the same as fruits and vegetables. Since it is harmless, its use may be continued for months. It is useless taken at intervals of several days. In moderate or severe cases of constipation cascara-agar is given daily in doses of one to three heaping tablespoonfuls, best after meals, morning, noon, and night, or only at noon and night. As it is as dry as straw, it requires a vehicle, and for this purpose apple-sauce and mashed potatoes have both been found useful; it can be intimately mixed up with either. It may be taken into the mouth dry and be washed down with some liquid without mastication. When the constipation is somewhat obstinate, the effect of cascara-agar becomes apparent after a few days, the stools being evacuated more easily than usual and of a softer consistence. Until this end is attained, it may be necessary to use an enema in order to secure a bowel movement. When the remedy has produced its effect, the quantity to be taken should continue the same for eight to fourteen days. After that the patient should gradually reduce the dose, ascertaining by actual experience the quantity necessary each day to pro-

duce a soft and easy evacuation. This quantity, in the great majority of cases, is considerably less than the dose required at the start. Even in cases of obstinate constipation two tablespoonfuls a day will frequently be found sufficient; in many cases much less will do. The quantity that is found to be just sufficient for the purpose may be continued daily for months. The hopes entertained with regard to cascara-agar have been fully confirmed. Agar is also given in biscuits, being added to the flour before baking. Each biscuit should contain 5 grains of agar (see page 287).

While searching for something which would fulfil the above-mentioned indications for constipation, and having the idea that a copious supply of fat would necessarily have a stimulating effect, twenty years ago A. L. Benedict, of Buffalo, found that liquid petrolatum given by mouth rendered the feces comparatively soft, while lubricating the intestinal mucous membrane. Liquid petrolatum is a clear, bland, neutral oil obtained from petroleum after the more volatile portions have been removed by distillation. It does not become rancid like the vegetable oils. Taken into the stomach, it passes through the whole intestinal tract unchanged; is not digested by any of the enzymes; and is thus able to exert to the full its emollient and lubricating action. It is absolutely non-irritating—a safe, mild laxative in tablespoonful doses three or four times a day. The oil can usually be found in the feces (see page 650). Characteristic of the best preparations of liquid petrolatum for internal administration, absolute freedom from taste and odor should be mentioned; also a sufficiently high degree of specific gravity to prevent premature passage through the bowel.

The "grape cures" enjoy more or less reputation, and in connection with their employment certain health resorts are particularly recommended (Meran, Bozen, Montreux, Dürkheim). The rule is to eat three to six kilos (six to twelve pounds) of grapes daily, the general diet meanwhile being rich in protein and non-irritating to the bowel. One kilo of grapes is eaten in the morning an hour before breakfast, and the remainder of the prescribed daily quantity an hour before dinner and supper respectively.

Massage of the abdomen and the intestine over the abdominal walls is fully described in Chapter X on Massage. It is very useful as an aid to the dietetic treatment. Parallel kneading of the abdomen with both hands is of great assistance (Fig. 102). The patient assumes the dorsal decubitus on the massage bed; the legs are stretched, slightly raised on a cushion in order to relax the tension of the abdominal wall. The physician stands at the patient's right side, making kneading movements over the anterior and lateral part of the abdominal wall in transverse direction; the hands work in opposite directions—the right hand from right to left, and the left hand from left to right, and then *vice versa*. In the transverse movements the right hand gradually rises from the symphysis to

the umbilicus, the left moving from the ensiform process down to the umbilicus, then both take the opposite direction. Swedish manipulation, rectal massage, rectal tampons, and electricity may also be employed advantageously (see pages 228-230).

Abdominal massage, calisthenics, regulated exercise, running, climbing, walking, rowing, swimming, riding, tennis, golf, or any other muscular exercise that seems advisable should be ordered for the patient of sedentary habits, and it must be urged on him that if the habit of constipation is not cured the future promises imperfect action of the liver, nervous irritations, early cardiovascular disease, and arteriosclerosis.



FIG. 102 -Parallel kneading of the abdomen with both hands for constipation
(Zabludowski-Eiger.)

Marked constipation is quite frequently seen in individuals who take a great deal of exercise and do much work. Boas has called special attention to the fact that good results are often to be attained after the "rest" and "recumbent" cures. Systematic home gymnastics are very useful, especially the exercise and strengthening of the compressor muscles of the abdomen, which is accomplished by repeatedly changing the posture from lying to sitting without the assistance of the arms. The posture during defecation is likewise of great importance. The position with elevated knees is the one most appropriate, because with it the abdominal pressure can be exerted most powerfully.

Should gastroenteroptosis be present, the application of a well fitting abdominal supporting bandage often brings about a prompt evacuation of the bowel. (See Chapter XXX on Gastroenteroptosis.)

Especially in cases of neurasthenia, hydrotherapeutic procedures, both general and local, may be employed which improve the appetite

and the general state of health. They stimulate peristaltic movements when applied locally upon the stomach in the form of the cold Scotch douche, cold towels, and cold sponge baths. The beneficial effect of the ether spray is well known. Twice a day 100 Cc. (3ij) of pure ether is sprayed on the skin of the abdomen. The cold developed acts as a strong stimulant to the abdominal walls and increases peristalsis. The local application of the electric cold-air douche has recently been recommended (Fig. 103, page 671).

Should these measures, with diet and agar, fail, the treatment by enemata must be resorted to. This has been fully described in Chapter XI. Generally speaking, enemata are indicated in dyschezia; the fecal mass lies in the rectum and cannot be forced out. They act by mechanically distending the rectum. Preference is to be given to the small "retention enemata" and to Fleiner's oil enemata (see page 223). In some instances the "paraffin cure" of Lipowski and carbon dioxid or bile enemata may be tried (see page 226).

There are no contra-indications to duodenal lavage in the treatment of atonic and spastic constipation. The whole intestinal canal can be thoroughly evacuated and a recovery brought about by this method of treatment (see page 105).

Medicinal treatment of chronic atonic constipation becomes justifiable only when the therapeutic measures mentioned fail to cure. The medicaments used for this purpose have been fully outlined on pages 282 to 288 (Chapter XIV). When prescribing purgatives, however, it should be the endeavor to secure results by the use of the smallest effective dose possible of the mildest drugs, such as castor oil, cascara sagrada, tamarinds, and phenolphthalein. Attempts should constantly be made to discontinue the purgatives entirely. When catarrh of the stomach or intestine coexists with constipation, Carlsbad salt or magnesium oxid should be administered. Podophyllin increases peristalsis and is at the same time a good biliary stimulant.

Good results are reported from yeast, taken once or twice a day, according to the number of bowel movements caused. The ordinary tin-foil yeast cake works almost as well as brewer's yeast. The amount administered should be about five-eighths to three-quarters of a cubic inch, dissolved in half a glass of water. This dose should be taken twice a day.

Mention has been made in Chapter XIV (Medication) of the subcutaneous injection of aloin, apocodein, eserine, eumydrin, and atropin. Magnesium sulphate can be administered subcutaneously in a dose of 0.5 to 5 Cc. (8 to 80 minims) of a 25-per-cent. solution. One cubic centimeter (15 minims) is the average and the most effective dose. The injections can be made in the buttock or under the skin of the abdomen, with the usual aseptic precautions. They are given daily until regular evacuations are established.

A yellow hygroscopic powder of crystalline appearance has been extracted from the bark of *Cascara sagrada* to which the trade name "peristaltin" has been given. It is readily soluble in water and can be given subcutaneously. The contents of one or two ampoules of 0.5 Gm. ($7\frac{1}{2}$ grains) each are injected in the course of twenty-four hours. An aqueous extract of senna is dispensed under the trade name "sennatin." The intramuscular dose is 1 to 3 Cc. (15 to 45 minims). The hypodermic injection of 2 to 4 Cc. (30 to 60 minims) of pituitary extract will stimulate the atonic and distended bowel when other measures fail; it is necessary to give larger doses than those needed to promote uterine contraction in labor; the 2-Cc. dose may be repeated, if necessary, in two or three hours.

An Italian method should be mentioned, the endermic administration of croton oil; a mixture of 6 to 10 drops of croton oil with 15 to 20 Cc. (3iv-v) of olive oil is rubbed into the skin. The results are said to have been satisfactory.

Primary and secondary atonic constipation may be treated by the drinking of mineral water. The secondary constipation that is found in connection with gastric and intestinal catarrh, hyperacidity, hemorrhoids, and liver derangement, is most favorably influenced by these drinking cures, particularly since the treatment influences the cause, and not merely the symptom constipation (see Chapter XII).

The most recent intravenous remedy for stimulating peristalsis is a hormone placed on sale under the trade name "hormonal." It was discovered by Zuelzer. Injected intravenously, it induces strong peristalsis. This hormone is extracted with normal saline solution or diluted hydrochloric acid from the mucous membrane of the stomach of an animal killed while the process of gastric digestion is at its height; the albumin of the extract is removed by alcohol. Small amounts can also be obtained from the proximal portion of the mucous membrane of the duodenum. This hormone is also obtained from the spleen; the quantities contained in this organ are so large that at present nearly all the available supply for therapeutic use is obtained from this source.

Zuelzer now recommends the treatment of chronic atonic constipation with intravenous injections of peristalsis hormone, 40 Cc. (3x) for adults and 20 Cc. (3v) for children. This is usually followed by a slight rise in temperature (to 100° F.), a feeling of warmth, some lassitude, and transient headache. No other evil effects whatever have been noted in consequence of these injections. The action of the remedy is often apparent after a few hours by the manifestation of increased peristaltic activity. The injection is best given in the morning. A single injection is sufficient to establish permanent results. It is advisable to follow the injection with one dose of castor oil. The latter serves as a lubricant while the hormone induces the normal peristaltic action.

Cleansing, glycerin, oil, paraffin, carbon dioxid and bile enemata are valuable measures in all forms of constipation (see pages 220-226).

Surgical Treatment.—Inflamed pericolic membranes forming bands and adhesions are causative factors in chronic obstipation. These membranes commonly cover the ascending colon, but may also involve the transverse and the descending colon. They can usually be easily stripped off from the intestine, leaving a clean smooth surface. The adventitious membrane is frequently as delicate as a spider web; Jackson has admirably described it in detail. These cases always require surgical intervention.

Stasis of the large intestine is a condition found in every case of chronic obstipation. Lane believes this stasis produces a toxemia which is responsible for many general nervous and debilitated conditions. When these cases have failed to respond to ordinary treatment, Lane treats them surgically by dividing constricting bands and adhesions, straightening kinks and angulations. In a considerable number the relief has been only temporary, obstruction recurring sooner or later. In such cases he performs lateral anastomosis between the ileum and the sigmoid, and later divides the ileum completely and implants it into the sigmoid; after this he removes the cecum and entire colon together with part of the sigmoid. In many cases he is able to relieve the toxemia and intestinal stasis by this procedure. (See Chapter XXXIX.)

SPASTIC CONSTIPATION.

In spastic constipation the retardation in the evacuation of the bowel is induced by a spasm (enterospasm) of a few isolated loops of the intestine (see page 781). This spasm is brought about by an increased irritability of the vegetative nervous system (see page 387), which may be due to neuropathic conditions associated with diseases of the abdominal viscera or pelvic organs, or to vagotonia or sympathicotonia associated with neurasthenia or hysteria.

The normal function of the intestine depends upon the innervation supplied by the two opposing systems of nerves, the vagus acting as the motor while the splanchnics are inhibitory. In a general way we call the two systems, which are so wonderfully well balanced, the vegetative nervous system. Aside from individual variations which may take place under normal conditions, the several organic functions may undergo far-reaching changes through excess of tonus on the part of one or the other of these sets of nerves. Every increased stimulation of the vagus exerts an increased influence on the activity of the muscles of the intestinal canal. Excessive stimulation, or vagotonia, induces muscular spasm of the small intestine, contraction of the colon, and other phenomena.

Spasms of the colon are observed by means of palpation and roentgenographic examination. They occur most frequently at the transverse colon, hepatic and splenic flexures, sigmoid, rectum, and anus. Spasm of the large intestine is important as the basis of spastic constipation, as distinguished from the atonic form. The characteristic symptoms of spastic constipation are delay in the fecal discharge, and an intestinal colic which usually precedes the defecation. In such cases there are various degrees of abdominal pain, with or without meteorism, which may affect the entire abdomen or only certain portions of the intestine. These pains are often continuous for hours, and finally terminate with the occurrence of defecation, which is often very voluminous. It is frequently possible to palpate the tender contracting intestinal loops, particularly those of the descending colon and the sigmoid flexure. There is frequent desire for stool, with incomplete evacuation. The dejecta are small-calibered, pencil- or ribbon-shaped, due to spasm of the sphincter. Spasm is associated with atony of the distal segments of the colon, and hypermotility and normal or reduced tonus of the proximal segments. Spastic constipation was once regarded as a separate phenomenon, but a more detailed study of the functional disturbances of the intestine has altered this view. Roentgenographic examination has revealed an increased physiologic distribution of the movements of the haustra, and hypermotility with disordered movements in the fecal current. Spastic constipation is not to be understood as a mechanical obstruction to the passage of the feces through the intestinal canal; if it were this, hypertrophy and distention of the sections involved would result. High-grade spastic contraction constitutes no hindrance to the forward movement of the intestinal contents. Not infrequently spastic and atonic conditions of the colon alternate or occur together, as is evidenced by the fact that the retention of feces in the ascending colon is accompanied by excessive tonus of other sections, such as spasms at the beginning of the transverse colon. When functional constipation varies in form, passing from one form into another, the movements of the colon presenting different conditions at different times, it may be that we shall have to unite these several manifestations into one group as spastic constipation (Plate XX, Fig. 2).

Spastic phenomena of the colon, with or without constipation, occur more frequently in women than in men, probably because of social conditions, the nervous status in the better situated classes, and the causal relation between intestinal function and diseases of the female genital organs. This is the case especially in pronounced neuropathic individuals who are subject to organic disturbances of the gastro-intestinal tract. In the neurasthenic, functional disturbances, among them constipation, usually arise without spastic change of the intestine.

The most varied conditions of the stool coexist with spasms of the colon and of the rectum, as to form, consistency, and frequency. In spasms of the colon with prolonged haustral segmentation, the fecal matter is in the shape of irregular balls, while in proctospasm it is cylindrical or ribbon-like.

There seems to be no definite correlation between tonic contraction and constipation; there may be a normal or a diarrhetic stool or *spastic diarrhea* in cases of pronounced spasm of the intestine. The test-diet stool in these cases is quite normal.

By regularly palpating the abdomen and by roentgenologic observation, strongly contracted sections of the colon may be located, especially of the sigmoid flexure and the descending colon. It is easy to distinguish between contracted and filled coils of the intestine by examining the patient repeatedly and at various times of the day. The tonus of the anal sphincter shows the presence of a proctospasm, which accompanies spastic contractions of the colon. The characteristic physical sign of this disease is found upon exploration of the rectum, which, instead of being filled with feces, as is common in atonic constipation, fits closely around the examining finger (proctospasm), almost like the finger of a glove (see page 848).

A phenomenon accompanying spastic conditions of the intestine and spastic constipation is colitis in various forms. Enteritis membranacea (see Chapter XXXVI) may be considered in this connection, for it is marked by spastic, cord-like contractions in large or small sections, especially of the sigmoid flexure, which can be palpated and roentgenographically proved during the colic as well as in the periods free from attacks (see page 140). Enterocolitis leads to colic and other spastic phenomena.

Treatment. This variety of chronic constipation should also, if possible, be treated only by means of dietetic measures. The diet should be given with a view to producing a chemical stimulation. The leading points of such a chemically active diet are elucidated on page 185 (Chapter VII). The following diet may be given:

EARLY:	Arising in the morning } to 1 kilogram (1 to 2 pounds) of grapes, watermelon or oranges
BREAKFAST:	Sardines in oil; tea with milk; white bread, butter (abundantly), honey or jam
NOON:	No soup. Meat; boiled vegetables; stewed fruit; egg with fruit juice; white bread, butter, honey, grapes, oranges, dates, figs, one glass of sweet cider
EVENING:	Cold meat or bacon - mashed vegetables with egg, white bread, butter, cheese, grapes, tea with whipped cream
10 P M	Grapes or fruit marmalade

Large quantities of fat in the shape of oil, butter and cream can be highly recommended, and also act beneficially upon a possible concomitant hyperacidity. The laxative diet may be assisted by the use of agar or cascara-agar.

Mechanical treatment is contra-indicated in spastic constipation. The intestine must be kept as quiet as possible; forced bodily exertions and gymnastics are therefore to be prohibited. Favorable effects are often observed from a food cure—hyperalimentation (see page 569). Massage of the abdomen and rectum is not permissible.

In regard to *electric treatment*, weak galvanism of the abdomen may perhaps be allowed. Sedative hydrotherapeutic measures, both general and local, may, however, be used freely. Withal, the fundamental disease, the nervous condition, should be improved. In France the application of the electric hot-air douche (Fig. 103) to the abdomen is highly recommended. This douche acts, on the one hand, by its warmth, which may be raised to a considerable height; on the other hand, by its mechanical effect, somewhat like a very light effleurage.

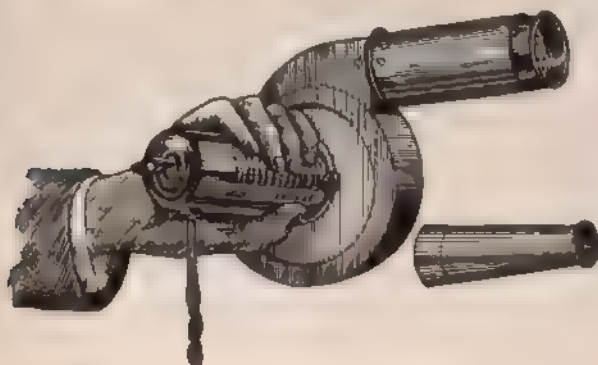


FIG. 103.—Hot- and cold-air douche.

Should these measures be found inadequate, enemata must be resorted to. Fleiner's oil cure is particularly advisable in cases of spastic constipation. For insertion into the rectum a flexible tube is used which is large enough to allow of the easy injection of the oil. This is connected by means of a soft-rubber tube with a syringe containing about twelve ounces of pure olive oil. The patient is to lie on his back, with the pelvis elevated; the tube is then gently introduced into the anus, and the oil is slowly and gradually allowed to flow. Usually twenty minutes is necessary to complete the injection. The tube need not be introduced high up into the rectum; if the patient is in proper position the oil will be carried up. Given with care, the injection of the oil produces no sensation. The patient may feel a desire to pass gas, but there is no pain if good oil is used. The injections are repeated daily for three or four days, then the amount of oil is decreased to eight ounces. The interval between injections may be lengthened as the patient improves. At first it may require a few days before the feces are

entirely evacuated. After this the comfort of the patient is much greater and the evacuations easier. The oil should be given at bedtime, slightly warmed, and retained all night if possible. Later it will be found that the injection of an ounce or two at bedtime will produce a satisfactory movement of the bowels in the morning (see page 223).

Purgatives, likewise glycerin enemata and glycerin suppositories, are to be strictly prohibited. The antispasmodic medicaments are preëminently indicated in cases of spastic constipation—as opium, extract of belladonna, atropin, and eumydrin; they may be administered by mouth or in the form of suppositories (see page 271). The dose of eumydrin is 20 drops of a 0.1-per-cent. solution three times a day. Belladonna inhibits the vagotonia, and in that way is valuable in the treatment of spastic constipation. It is also a stimulant to peristalsis. Papaverin hydrochlorid relaxes smooth muscle in general. It is most effective in spastic conditions, while it does not interfere materially with the normal movements of the intestine. Its toxicity is low, and neither tolerance nor habituation from its use has been reported. It is especially valuable in all kinds of gastric and intestinal spasms and in biliary colic. Benzyl benzoate is equally efficacious (see page 276). Preparations of bromid may be given to quiet the nervous system. The reader is referred to Chapter XIV on Medication.

Many clinicians have obtained good results by dilatation of the anal sphincter. This may be accomplished by the use of the rubber dilator (Figs. 153 and 154), bougies, or the speculum. Divulsion can also be performed under an anesthetic (see pages 838-841).

FRAGMENTARY CONSTIPATION.

Boss has described this affection and named it fragmentary evacuation of the feces. The patients have regular and unaided, but incomplete, movements of the bowels, with repeated desire to defecate. They will endeavor to have a movement every two or three hours, and, notwithstanding considerable effort, are able to evacuate each time only a very small quantity. This is not followed by a sensation of relief, but, on the contrary, by tenesmus and a feeling of fulness in the abdomen. This disturbance in the fecal evacuation is particularly frequent in men, and is due to a relaxation of the lower sections of the large intestine, perhaps only of the rectum.

Treatment.—The treatment may be gathered from what has been already said. It consists particularly in diet, with the addition of cascara-agar. When the subjective discomforts are urgent (tenesmus), anodynes must be used. The relaxation of the rectum may be corrected, to a considerable extent at least, by intrarectal faradization and massage. (See Chapter XI.)

CHAPTER XXXVIII.

CHRONIC DIARRHEA.

GASTROGENIC DIARRHEA; INTESTINAL FERMENTATIVE DYSPEPSIA; NERVOUS DIARRHEA.

FOR much of our recently acquired knowledge of the cause and treatment of chronic diarrhea we are indebted to the systematic examination of the feces, as inaugurated by Adolf Schmidt. In the medical mind chronic diarrhea was at one time synonymous with intestinal catarrh and nervous diarrhea, but the systematic examination of the feces and the proper testing of the activity of the intestine has shown that many other conditions must be included in the meaning of the term. We are now in a position to differentiate, aside from the diarrhea of chronic intestinal catarrh, the following forms of diarrhea:

- I. Gastrogenic Diarrhea.
- II. Intestinal Fermentative Dyspepsia.
- III. Nervous Diarrhea.

GASTROGENIC DIARRHEA.

A large proportion, probably the majority, of all cases of chronic diarrhea are due to disturbances in the gastric function. It has been shown by Adolf Schmidt that raw or smoked connective tissue cannot be digested except by normal gastric juice. The intestine does not share in the digestion of such connective tissue, but it does dissolve boiled connective tissue. When patients affected with subacidity and achylia gastrica are fed with raw or smoked connective tissue, the material reappears completely unaltered in the feces. If, therefore, Schmidt's test diet (page 112) be given to a patient with achylia gastrica who shows no intestinal disturbance whatever, a stool is obtained which differs from normal feces only in the fact that it contains the connective tissue of the test-diet meal in an unaltered condition. This finding characterizes 99 per cent. of all cases of achylia and subacidity; an isolated exception is occasionally noted. Some cases in which the connective tissue reappears in the stool have an altogether normal secretory condition of the stomach, and others even hyperacidity. Conversely, there are cases of achylia in which the stomach is capable of digesting connective tissue, as shown by an examination of the feces. These exceptional cases are difficult to explain. They seem

to show that there may be a disproportion between the hydrochloric acid secretion and the production of pepsin. It has been proved that deficiency of pepsin may exist in hyperacid conditions, and that, on the other hand, in spite of the absence of hydrochloric acid a sufficient quantity of pepsin may be present.

Einhorn and Laporte have made a tabulated report on the amount of pepsin found in 110 cases of various stomach disorders. They conclude that in cases where hydrochloric acid is increased the pepsin units remain the same. The action of the pepsin is markedly rapid when hydrochloric acid is increased. In cases with diminished secretion of hydrochloric acid, pepsin production is also less. In cases of entire lack of secretion of hydrochloric acid, pepsin is always present, but possibly only a trace of it.

It is perhaps the pepsin which is the absolute essential in the digestion of connective tissue. In cases of hyperacidity with incomplete digestion of connective tissue, hypermotility of the stomach and pyloric insufficiency must be kept in mind; the stomach may be unable, for want of time, to digest the connective tissue. In many cases, however, no cause can be discovered for the reversal of the rule stated by Schmidt. It is possible that a rôle at present unknown is played by the altered conditions of the gastric juice. There is no doubt that slight alterations in the work performed by the stomach are enough to interfere with the digestion of connective tissue.

The reappearance of macroscopically recognizable remains of connective tissue (see page 119) in the feces after the test diet proves conclusively that the stomach has been unable to perform its proper work. The connective-tissue test, therefore, is an exceedingly fine one for ascertaining the functional condition existing in the stomach. It is a diagnostic sign of the presence or imminence of gastrogenic intestinal disturbances. The intestinal symptoms may make their appearance in the form of vague subjective sensations or momentary irritations of the bowel which, without the test diet, would hardly be thought worthy of serious consideration. In atonic conditions of the stomach, with decomposition processes going on, though there is hydrochloric acid in the gastric juice, the test connective tissue is occasionally found in the feces undigested.

In answering the question tentatively, "How is it possible that such disturbances of the stomach can lead to diarrheic conditions?" it is well enough to remember that formerly the greatest importance was ascribed to the absence of the antiseptic action of the gastric hydrochloric acid; but this is certainly not the only deciding point, for the mechanical irritation of the undigested connective tissue on the intestinal mucous membrane itself, especially when often repeated, is apt to induce liquidity and frequency of fecal discharges. On observing how large quantities of connective tissue and muscle remnants (see page 120) are excreted in cases of achylia,

particularly after eating rare meat, it is easily understood that the oft-repeated transit of such masses of connective tissue must of necessity greatly irritate the delicate mucous membrane. Of probably greater importance, however, is the fact that coarse connective-tissue remnants constitute an ideal culture medium for all sorts of bacteria to multiply in the stomach. Putrefactive and fermentative organisms are swept into the intestinal canal, where they develop abundant colonies, infecting the gut and leading to disintegrating processes. Gastrogenic diarrhea is probably most frequently brought about in this manner, and in such cases it is by no means rare to find among the bacterial growths of the feces long bacilli, sarcinae, yeast, and many other specimens derived from the stomach. Furthermore, it must be considered that the increased demands made upon digestion in the small intestine when the stomach is not functioning properly lead gradually to intestinal insufficiency.

Symptoms.—There is at first an irregularity in the movement of the bowels, followed by an occasional attack of diarrhea, to which, as a rule, no particular attention is paid. The diarrhea gradually becomes more frequent until no solid feces are excreted. The laxness of the bowels constantly increases until occasionally the gravest symptoms of intestinal disease follow slight derangements of the stomach. The mucous membrane of the intestine is unable to endure, for any length of time, such abnormal conditions. The continuous irritation finally leads to inflammation, resulting in a secondary catarrh of the small intestine, and sometimes also of the large intestine (see page 644). This disease of the intestine may progress while the primary affection of the stomach improves, until it finally appears to be an entirely independent disease. For this reason the gastrogenic origin of chronic diarrhea is frequently entirely overlooked. But examination of the feces after the test diet (see Chapter IV) gives positive information; the appearance of the feces is not always uniform, but in all cases of gastrogenic diarrhea there will be traces of undigested raw or smoked connective tissue after ingestion of this material in the test meal. This finding indicates that a disturbance of the stomach is either present or imminent, and an examination of the stomach contents after an Ewald test breakfast will, in the majority of the cases, be sufficient to confirm the diagnosis.

Other findings in the feces will vary according to the gravity of the affection of the intestinal mucous membrane. We may have the signs of inflammation, namely, mucus, tendency of the liquid stools to processes of putrefaction with the development of odors, and, in rare cases, pure carbohydrate fermentation. Furthermore, under certain circumstances, disturbances in absorption may indicate the degree of the affection of the mucous membrane of the small intestine. The chief point, however, is the discovery of connective tissue in the feces (see page 134).

Treatment.—The treatment of gastrogenic diarrhea consists in energetically dealing with the diseased stomach. Get the stomach right, and the intestinal difficulty will frequently disappear without further treatment. The success of such therapeutics, which in most of the cases consists in counteracting the deficiency in hydrochloric acid, is certainly surprising. An endeavor should always be made to treat the stomach in obscure cases of chronic diarrhea in which direct examination of the stomach contents shows both secretion and motility, but in which the feces under the test diet point to the stomach as the seat of the disease. If the feces contain either connective tissue or certain special bacteria, of presumably gastric origin, a trial of the stomach treatment should be made at all events. One clear indication is to eliminate rare or smoked meat containing connective tissue from the diet of patients suffering from either gastric or intestinal disease. The diet must always be adapted to both the gastric disturbances (achylia, catarrh) and the temporary condition of the intestine (catarrh of the small or large intestine, irregularities in absorption, putrefaction, carbohydrate fermentation). A constipating diet is always indicated (see page 172). When hydrochloric acid is absent it should be given freely as a medicament, together with pepsin (see page 258).

Owing to the absence of hydrochloric acid from the stomach, there is no stimulus for the production of pancreatic juice, and consequently the digestion lacks this latter important element also. This explains the efficiency of hydrochloric acid given to patients with gastrogenic diarrhea. The acid cannot be given in amounts sufficient to replace the missing gastric secretion, but the amount ingested is sufficient to start pancreatic secretion.

Examination of the stomach contents after an Ewald test breakfast will decide whether the presence of connective tissue in the feces is due to hyperacidity or to achylia. The medicinal treatment for this functional derangement must be with either the acids or the alkalis. The best combination of alkalis in gastrogenic diarrhea with hyperacidity is the following:

	Gm. or Co.
R Creta præparata,	
Calci carbonatis præcipitati,	
Calci phosphatis præcipitati . . .	ss 30 0 3j
Misce	
Sig. —Teaspoonful in half-glass of water immediately after meals.	

Bismuth subnitrate or bismuth subcarbonate may be added to the above combination (see page 265).

For its beneficial action on the mucous membrane of the intestine, no drug is so satisfactory as bismuth. When an astringent action is required, subgallate of bismuth in the dose of 0.3 to 0.6 Gm. (5 to 10 grains) every four hours can be given; for a more

stimulating effect, the subnitrate of bismuth in doses of 0.6 to 1.3 Gm. (10 to 20 grains) three times a day; as an antiseptic, the salicylate of bismuth in doses of 0.3 to 0.6 Gm. (5 to 10 grains) every four hours. The sedative action of opium, uzara, bolus alba, tannic acid, and their preparations, has been fully described on pages 274 to 279. Should there be atony and pyloric insufficiency, strychnin sulphate should be given in full doses until the physiologic action of the drug is secured (page 407). It is well to begin with 0.001 Gm. ($\frac{1}{60}$ grain) three times a day and increase the dose until 0.003 Gm. ($\frac{1}{20}$ grain) is being taken. Should there be no twitching of the muscles, the dose can be increased to 0.006 Gm. ($\frac{1}{10}$ grain), and gradually to 0.01 Gm. ($\frac{1}{10}$ grain) if necessary. Gastrogenic diarrhea associated with pyloric insufficiency responds quickly to the strychnin treatment, providing the perverted stomach function be rationally treated at the same time. Strychnin is not contra-indicated in gastrogenic diarrhea associated with pyloric insufficiency. Pfaff and Nelson have observed the peristalsis in rabbits and cats under the Roentgen ray and when the intestine was bathed in saline solution. They found that nux vomica did not increase peristalsis. Diarrhea can often be controlled by allaying the irritation of the gastrocolic reflexes. This is best accomplished by anesthetizing the gastric mucous membrane by administering 0.008 to 0.015 Gm. ($\frac{1}{8}$ to $\frac{1}{4}$ grain) of cocain a quarter of an hour before meals for three or four days.

When patients are anemic, an iron astringent will sometimes act favorably upon the diarrhea and the anemia at the same time. The sulphate of iron in capsule, in a dose of 0.2 to 0.25 Gm. (3 to 4 grains) three times a day, after meals, can be prescribed.

Secondary intestinal catarrh is to be treated appropriately (see Chapter XXXV). Regularly continued lavage of the stomach must not be forgotten, as it is often effective even in cases in which all other gastric treatment has been unsuccessful. Cases of chronic diarrhea of several years' standing can often be cured completely by lavage of the stomach.

INTESTINAL FERMENTATIVE DYSPEPSIA.

A considerable number of people are met with who suffer from frequent abdominal pains, noises in the abdomen (borborygmi), flatulence, chronic meteorism (distention), disturbances of the appetite, general discomforts, and frequent liquid or semisolid stools which are evacuated two to four times daily. Notwithstanding all this, the patients do not appear to be abnormal. When, however, they are placed on a test diet, they pass a stool which is either thin or semifluid, yellowish or bright yellow in color, and large in volume; it is pervaded with numerous small bubbles of gas, and has an intensely sour odor even when fresh. When the feces

are carefully examined with the unaided eye, no remnants of protein bodies are found; connective tissue and meat are seen to be perfectly digested and the quantity of fecal fat not at all excessive. On the other hand, remnants of the mashed potatoes ingested are found in huge amounts, partly in the shape of coarse, cohering, sago-like particles, and partly in the form of single potato cells. On microscopic examination after the addition of concentrated Lugol solution, it is seen that the potato cells are not broken up, and that the starch enclosed in them is present in an unchanged condition. In addition, large numbers of loose starch granules are seen which have lost their cellular coating and which, on the addition of iodine, become intensely blue in color. Besides, there are usually present large numbers of the iodine bacteria—mention of which is made in Chapter IV on Examination of the Feces.

Normal red coloration of the feces is shown by the "sublimation test" (Plate VII, *a*). When the feces are kept in the incubator enormous quantities of gas are generated and the previous slight acidity becomes marked, the feces emitting a penetrating sour odor. (Plate VIII, *b*.) Occasionally the freshly passed stools are quite normal in shape, and only careful microscopic and macroscopic inspection will excite suspicion. Of all the nutrients ingested, only the carbohydrates are poorly or not at all digested. These undigested carbohydrates are subjected to the action of the fermentative bacteria (to which class preeminently belong the iodine bacteria); they undergo fermentation, becoming strongly acid, evolving a great deal of gas, and giving the stool a characteristic putty appearance (see page 134).

This specific disease-picture is by no means rare, and many patients are met with who have suffered for years from these exceedingly annoying symptoms. In such cases a single analysis of the stool with the test diet will clearly determine the pathologic condition. When fermentation processes of this kind persist for a long time, it is of course possible that the intestinal mucous membrane may be greatly irritated, and a secondary catarrh induced which naturally aggravates the disease. Schmidt believes the fermentative dyspepsia is caused by a derangement of the secretion of the small intestine, which is normally a powerful amylolytic ferment, because the other digestive secretions (gastric juice, pancreatic juice, and bile) perform their functions perfectly. In non-complicated cases of intestinal fermentative dyspepsia no signs of any organic intestinal disease have been found, even on postmortem examination. There is another explanation, and Schmidt admits its possible correctness; it is that the ferment in the small intestine which breaks up the cellulose may be absent. Lohrlich has quite recently demonstrated that the existence of such a ferment must be assumed as an indisputable fact. It is quite easy to understand that when the ferment does not dissolve the

cellulose coverings which enclose the starch granules of the potato cells, the starch necessarily must remain undigested. Lohrlich has also been able to demonstrate that patients with intestinal fermentative dyspepsia excrete on a test diet much more cellulose than the normal person. In confirmation of the view that the "cellulose ferment" is at fault, the fact may be cited that well-dextrinated flour is more fully used up and considerably better borne by this class of patients than are potatoes (see page 61).

Intestinal fermentative dyspepsia is, no doubt, in many instances a primary disease, due to disturbance of the functions of the small intestine, as stated; but it may also occur secondarily to chronic catarrh of the small intestine, hyperacidity, achylia, or gastrogenic diarrhea.

Treatment.—The treatment of these conditions, particularly of the primary disease, is clearly indicated by the findings of fecal examinations, and consists chiefly of purely dietary measures. The diet should be such as to counteract the processes of fermentation; the details are fully given on page 181 (Chapter VII). At first a pure protein-fat diet should be maintained, with complete exclusion of carbohydrates, by which at one stroke the discomforts, borborygmi, flatulence and diarrhea are removed, and the stools immediately become firm in consistence. This having been accomplished, well opened-up cells of carbohydrates, infant flours, dextrinated flour, toast, zwieback and crackers are to be slowly added. Later, and very gradually, farinaceous products richer in flour may be allowed, and then the vegetables. Potatoes must be prohibited for a long time, and also all those species of vegetables which contain large proportions of starch, cellulose and sugar, as turnips, carrots and celery. When the symptoms are more acute it may be necessary to initiate the treatment with rest in bed and the application of either moist or dry heat to the abdomen.

Irrigation of the intestine with normal saline and antiseptic solutions may be accomplished by direct instillation into the duodenum by means of the duodenal tube (see Chapter III).

Pure oxygen has been used as an intestinal antiseptic. Schmidt introduces as much as 2 to 4 liters of oxygen through an Einhorn duodenal tube (Fig. 11). He has found it valuable in the treatment of both pathologic putrefaction and fermentation of the intestinal contents. When the gas is introduced slowly it never inconveniences the patient. Colic or peristaltic unrest does not occur. In one or two hours after its introduction odorless flatus passes the anus, showing that the oxygen has passed through the whole length of the large intestine. The inflation is given at first twice daily and, as improvement ensues, once a day (see page 105).

In secondary fermentative dyspepsia the fundamental disease of the stomach or intestine must be treated in preference to everything else.

NERVOUS DIARRHEA.

As already stated, systematic examination into the functional activity of the intestine and the composition of the feces has rendered possible a sharp and accurate differentiation between diarrhea occurring in the course of intestinal catarrh, that of gastric origin, and that due to fermentative dyspepsia. This methodic arrangement has essentially reduced the number of diarrheic varieties which were formerly included in the class of nervous diarrheas. It is now recognized that a large number of cases of so-called nervous diarrhea are due to other etiologic factors. Yet there undoubtedly exists a real nervous diarrhea which runs its course without perceptible anatomic lesion or functional derangement of the intestine.

Two forms of nervous diarrhea are now recognized—a psychogenic and a reflex variety. Mixed forms combining both of these are also described. In the first variety the urgency for immediate defecation with diarrheic frequency may be induced by psychic excitement, psychic events, often even by the mere contemplation of some possible event that may be either pleasant or disagreeable in character. The best example of this is the diarrhea provoked by fear. Reflex diarrhea, on the other hand, is induced by bodily stimuli. Many people have an attack of diarrhea after partaking of some special articles of food or drink, or when the food has some particular temperature. In other cases, again, affections of the genito-urinary tract and of other organs play a rôle. Certain irritations emanating from the skin may lead to diarrhea. The characteristic point about nervous diarrhea is that the psychic (mental) or bodily stimuli which evoke the diarrhea are always quite distinct and do not exceed a certain etiologic limit; in healthy individuals such stimuli would not be productive of diarrhea. The question therefore arises: What are the special conditions under which these by no means excessive stimuli are liable to give rise to diarrhea? Like all the other varieties of diarrhea, so with the nervous diarrheas, they are all dependent on a hypermotility of the muscle fibers of the intestine and an increase in the secretion of the intestinal juices. The irritative effect is probably due to inaction of cells or forces in the vegetative nervous system which normally inhibit the activity of the muscular and glandular elements of the intestine. It is possible that stimuli which normally pass off without producing any effect stimulate both the glandular and the muscular structures of the intestine in cases of nervous diarrhea. As, however, not every kind of stimulus causes diarrhea in such patients, but only one or several quite distinct stimuli, it follows that the conductivity for these special stimuli must be quite specifically facilitated, and this is made possible by means of distinct pathologic paths within the vegetative nervous system

(see page 387). This is, however, conjectural, as nothing really definite is at present known about these matters.

Symptoms.—Clinically, the course taken by nervous diarrhea is such that any age of life may fall under its pathologic influence. If nervous diarrhea has occurred once only, induced by any one kind of mental or bodily irritation, the disease, dating from that moment, may become chronic, often demonstrating its presence in a most unpleasant manner. At every recurrence of the particular slight irritation, symptoms of various kinds may make their appearance, such as abnormal sounds, painful sensations in the abdomen, and suddenly a liquid fecal evacuation. These conditions may attain so aggravated a stage that the patient finds it impossible, on account of them, to leave home. It will be readily seen how many disappointing situations, in regard to both social and business affairs, are liable to be occasioned thereby. The victims of nervous diarrhea frequently become confirmed neurasthenics. A preëxisting neurasthenia of course predisposes to the development of nervous diarrhea (see page 418).

Diagnosis.—The test-diet stool in cases of nervous diarrhea resembles very closely the recent contents of the small intestine, and when freshly evacuated does not show any signs of pronounced putrefaction or fermentation, neither does it contain any mucus. Microscopically, however, numerous food remnants may be seen which have escaped digestion in consequence of the increased intestinal motility. The stool never contains connective tissue (Plate VI). (See page 134.)

Apart from the quality of the feces, the previous history is of great importance, frequently explaining the kind of irritation and the slight intensity of it, and indicating the absence of organic diseases of the stomach or intestine and the existence of a nervous temperament.

Treatment.—A consideration of the foregoing statement makes it apparent that the treatment of diarrhea of this kind with astringents is both useless and wrong. On the other hand, the principal endeavor should be to treat the mind (psychic). To begin with, the patients must be assured that their digestive organs are anatomically normal; pessimistic moods must be alleviated, and the attention should be distracted from the digestive apparatus. To all this should be added a thorough hydrotherapeutic treatment, with the object of influencing the entire organism rather than the digestive organs alone. The full and half-baths, douches and frictions deserve particular consideration. An invigorating general treatment should be instituted. Simultaneously ferruginous and arsenical preparations should be prescribed. Arsenical mineral waters should be systematically taken. (See Chapter XII.)

Sojourn at climatic cure resorts of moderate altitudes (2000 to 3000 feet) is to be highly recommended.

No specialized rules can be laid down for the dietary treatment. In many cases it is quite useless to order a carefully selected diet. Some cases do well on a diet poor in residue and non-irritating in character. Experimentation is absolutely necessary. When the previous history shows that some particular article of diet induces diarrhea, this should be avoided. A lactovegetable diet is often very beneficial. A sudden total change in the diet frequently produces surprisingly good results. At other times a constipating diet is beneficial (see page 172).

All these measures can be followed up most satisfactorily when the patient is able to remain either at a sanatorium or in a private hospital.

The treatment of the acute diarrheic seizure, when the latter is very violent and accompanied by much pain, consists of rest in bed and the application of warmth to the abdomen. In such cases it will be found impossible to exclude opium entirely from the list of medicines (see page 274).

The general systematic treatment, as described in Chapter XIX on Nervous Dyspepsia, can be applied here. In case of enteroptosis the application of a suitable bandage will give great comfort. Subcutaneous injections of sea water in doses of 30 to 100 Cc. (3j iiij) every other day have a general tonic influence (page 423). The bromids and chloral in small doses may be necessary to relieve nervous irritability. The glycerophosphates of sodium and calcium, with lecithin, have been used with marked success. Menthol and thymol in small doses have been recommended. The intramuscular injection of iron, 0.05 Gm. (1 grain) of ferric citrate once a day, is indicated in anemic patients. This medication may be combined with the oral administration of glycerophosphate of sodium 0.1 Gm. (2 grains). Cacodylate of sodium can be given subcutaneously once a day, in doses of 0.05 to 0.2 Gm. (1 to 3 grains). (See page 581.)

Eppinger and von Noorden state that many cases of nervous diarrhea, and especially the diarrhea of Basedow's disease, respond quickly to the rectal administration of epinephrin. After the rectum is cleansed with an enema, 20 to 30 drops of a 1:1000 solution of epinephrin diluted with 10 ounces of warm water are injected into the rectum. No unpleasant subjective symptoms have been noted.

CHAPTER XXXIX.

INTESTINAL TOXEMIA, INTESTINAL STASIS, AND ILEAL REGURGITATION.

Intestinal Toxemia.—Intestinal toxemia is a form of blood poisoning induced by the absorption of toxins or microorganisms from a damaged intestinal mucous membrane. Any delay in the passage of the intestinal contents through the various segments of the intestinal tract exposes the patient to the danger of intestinal toxemia. The main fault is digestive—some disturbance, more or less severe and more or less prolonged, of the digestive processes. Of course digestion is in the main due to enzymes, but a small part of it is accomplished by bacteria; certain parts of the food which resist the action of enzymes would not be digested at all were it not for the microorganisms in the intestine. On the other hand, it should not be overlooked that the work of the intestinal bacteria is associated with the production of toxins against which normally the organism must protect itself. The decomposition products of bacterial activity are not all toxic, but in large amount they may have a decidedly toxic effect. Should the protective agencies of the organism, such as the neutralizing effect of the digestive juices upon the toxins, the antitoxic power of the hepatic cells, the detoxicating effect of the internal secretions (thymus, thyroid, adrenals) and of the epithelium, the excretion of intestinal toxins by way of the expiratory air, the urine and the feces—should these, for any reason, fail to act or act inadequately, intestinal toxemia would result. Bacteria and their toxins are believed to pass through the damaged mucous membrane into the wall of the intestine and induce inflammation, with the formation of pericolic membranes (see page 561), though no conclusive proof has yet been adduced to substantiate this view.

Intestinal Stasis.—The term "chronic intestinal stasis" as employed by Lane indicates such abnormal delay in the passage of the intestinal contents through a portion or portions of the gastrointestinal tract as to result in the absorption into the circulation of a greater quantity of toxic or poisonous materials than can be disposed of by the liver or other protective organs. He believes this condition is due to the upright position of the trunk, which induces a prolapse of the viscera (gastroenteroptosis, see page 561) and consequent faulty drainage. To resist this displacement, Nature reduplicates certain peritoneal tissues, which becomes firmer

and firmer until distinct bands are formed. As a consequence, under unusual or prolonged tension, kinking occurs (see page 561). The kinks occur at the duodenojejunal juncture, the terminus of the ileum, in the cecal region, at the hepatic flexure, the splenic flexure, and the colon-sigmoid juncture (Fig. 89).

Ileal Regurgitation.—When the ileocecal valve is incompetent, there is a backflow of fecal matter from the cecum into the ileum. This condition can be easily diagnosed by means of the Roentgen ray (see Plate XIX, Fig. 2). Much has been written in recent years on this condition. Many normal persons have ileal regurgitation; it does not do them the harm that some authors would lead us to expect. When the condition is pathologic, the symptoms of intestinal toxemia and intestinal stasis are present. Surgical measures are not necessary; the patients respond to the usual treatment of chronic intestinal stasis (see page 783).

Etiology.—The factors which contribute to the development of intestinal toxemia are manifold. Even when the condition of the digestive juices is entirely normal, frequent, large and albuminous meals may be abnormally decomposed. This danger is increased if there are secretory and motor disturbances of the stomach or intestine, as in achylia, gastric dilatation, pyloric stenosis, gastro-enteroptosis with kinks, constipation, catarrh of the small and large intestines, dilatation of the colon, intestinal stenosis, chronic appendicitis, and parasites. In these cases nutriment of normal quality in normal quantity may work harm. A further factor may be supplied by the weakening of the defenses of the organism, through infectious diseases, affections of the intestinal mucosa, hepatic insufficiency, anemia, or alcohol (see page 668).

Some authors believe that the theory of a bacterial cause of chronic intestinal toxemia is the most plausible. They assert that the colon bacillus cannot do any harm unless there is intestinal putrefaction or a denuded mucous membrane. Under such conditions the colon bacilli themselves become pathogenic by undergoing a sort of metamorphosis, with the liberation of toxins, the absorption of which causes many of the symptoms of chronic intestinal toxemia.

It has recently been shown by Kendall that when carbohydrates are present in the nutritive environment of most bacteria, the products formed are chiefly those which arise from the fermentation of the carbohydrates. When utilizable carbohydrates are not available, the bacteria must utilize the nitrogenous constituents of the medium. Under these conditions, nitrogenous waste products become relatively large and are more or less toxic. The colon bacillus grown in sugar-free nitrogenous media will produce indol, ammonia, hydrogen sulphide and the broken-down protein products, while if utilizable sugars are added to the media the same organism will not produce any of the products indicative of protein

break-down. Other microorganisms will form highly potent toxins when they develop in protein media, but form innocuous fermentation products when utilizable carbohydrates are added to the culture medium. It therefore seems logical to limit the production of these toxins by flooding the intestinal tract with large quantities of suitable carbohydrates to provide the necessary non-nitrogenous pabulum for the organisms in the intestinal canal.

An irritation anywhere in the intestinal canal will impede the circulation of blood at that point. The neutralizing alkalis are thus lessened, and the result is a dry, inflamed pathologic area. Here the absorption of toxins occurs which is responsible for so many symptoms.

There seems now to be little doubt that endemic goiter is due to an infection. The organisms reach the intestinal tract through the infected soil, water and food. Animal experimentation substantiates this view. It is also possible that the pathogenic agent may be one of the various organisms or toxins harbored in the tonsils, or in diseased sinuses or teeth (see page 290 and Plate XXIII).

Course of Intestinal Toxemia.—Even in normal digestion aromatic substances and ptomains are formed in the intestine, owing to the action of the bacteria upon the proteins. These substances are partly excreted in defecation. If the quantity increases, consequent diarrhea will hasten their expulsion. The residue will be neutralized by the mucosa, assimilated, passed through the liver and taken up into the circulation, where it will be definitely decomposed by the action of the internal secretions. The end-products will then be excreted through the skin, the lungs, and the kidneys. If, then, these products of normal digestion are toxic, the decomposition products of abnormal digestive processes must be so to a much greater extent. The most toxic products are always formed from the proteins, and it is these above all that cause intestinal toxemia.

Metchnikoff points out that the secretions of the bacteria differ with different food. If a little fecal matter be placed in two tubes, one of which contains chopped meat in water and the other chopped vegetables in water, the fluid in the first tube after two days becomes extremely poisonous to rabbits, while the fluid in the second is entirely harmless to them. The bacterial products are thus different in the two tubes, although the bacteria are derived from an identical source. Metchnikoff reports his own researches on the bacteria of putrefaction and their toxic products; in particular the anaerobes, *Bacillus putrificus* and *Bacillus perfringens*. He has demonstrated that these anaerobes are a source of toxemia against which the organism must struggle with all the means at its disposal. It is during the first few days of the development of these putrefaction anaerobes that their products are most intensely

toxic. This is interesting in view of the assertion so frequently made that putrefaction in the intestine is only the first stage of a process that continues outside the human body. He shows further the analogy between the proteolytic flora in the intestine and putrefaction in general, wherever encountered, and states that this constitutes a new argument in favor of the pathologic importance of the bacteria of putrefaction in the human organism.

Bacterial Growth.—Probably the most valuable means of determining the amount of bacterial growth in the intestine consists in directly weighing the bacteria isolated from the feces by the method of Strasburger. In this method the bacteria are separated from the other solid constituents of the feces by centrifugation. From one-eighth to one-fifth of the total dry weight of the feces is made up of bacteria, and in conditions of intestinal disease the proportion may amount to one-third or even more (see page 118).

Indican.—In the breaking up of protein by the putrefactive process, a number of substances which have a toxic and injurious effect upon the body are produced, and these are absorbed from the intestine. They all belong to what is technically known as "the aromatic series." The best known of these are skatol, indol, and phenol. These aromatic products of intestinal putrefaction have much to do with the production of pericolic membranes, bands and adhesions found in cases of chronic intestinal stasis. They are carried to the liver, where they combine with sulphuric acid, and are excreted in the urine as ethereal sulphates. An excess of ethereal sulphates in the urine becomes thus the measure and gauge of the degree of existing intestinal putrefaction. Indican in the urine has the same significance. The indol is rapidly absorbed from the intestinal tract and carried by the portal blood to the liver, where it enters into loose combination with liver cells; from this combination it is readily detached, to become united with sulphuric acid; before becoming thus united, however, it is oxidized into indoxyl so that, when united, it becomes chemically a potassium salt known as indoxyl sulphate of potassium. This substance is much less toxic than indol, finds its way into the blood, and is promptly excreted in the urine as indican. In early life the production of indol in the intestine is in general very slight; and there are some older persons also who, even while suffering from disorders of digestion, do not form indol. On the other hand, the production of considerable quantities of indol in the large intestine is a feature of many cases of intestinal putrefaction, and in some cases the quantity formed is large. That indol may be absorbed in considerable amounts is shown by the appearance of large quantities of indican in the urine of persons in whom the intestine contains large amounts of indol (Herter).

We cannot, however, depend upon the presence of indican alone in the diagnosis of intestinal toxemia. Indican in increased quan-

ities is usually present in the urine in acute and chronic gastritis, acute and chronic peritonitis, typhoid fever, dysentery, ileus, carcinoma, cholera, Addison's disease, disease of the central nervous system, empyema, gangrene of the lung, and all conditions where protein putrefaction is in progress. The finding of indican in the urine is not of itself sufficient to establish the diagnosis, but it has some value in connection with the other signs of intestinal toxemia.

Intestinal toxemia is possible without indican and with a perfectly healthy pancreas, or at least with one so judged to be by the complete digestion of nuclear tissue (see page 126). Those who believe that there can be no intestinal toxemia without indican in the urine will overlook many cases. Cholin forms the base of the lecithins which are abundantly present in various animal structures, but is in itself innocuous; it can, however, by the action of bacteria, be transformed into neurin, which is a highly toxic substance. Cadaverin and putrescin are bases and products of protein decomposition.

Types of Intestinal Putrefaction.—The variations in the clinical manifestations and pathologic accompaniments of chronic intestinal putrefaction suggest that the etiologic conditions vary in different patients, and Herter has suggested the three following types:

1. The Indolic Type, marked by striking indicanuria and probably due to members of the *Bacillus coli* group.

2. The Saccharobutyric Type, which seems to be initiated chiefly by the anaërobic forms. In its simplest examples there is very little indol in the gut.

3. A Combined Type, or cases combining the characteristics of groups 1 and 2.

Indolic Type. "In these cases the members of the *B. coli* group form indol in considerable quantities and they probably invade the small intestine in large numbers. The bacterial cleavages seem largely to replace normal tryptic digestion."

Saccharobutyric Type. "In this type the seat of the putrefactive process is the large intestine and lower ileum. It is due to the activity of the strictly anaërobic butyric-acid-producing bacteria. Although our study is not yet exhausted, it may be confidently stated that in many cases *B. aerogenes capsulatus* is largely responsible. With this form may be associated *B. putrificus* and possibly sometimes the bacillus of malignant edema, although often these forms are not found in cultures on any of the ordinary media.

"The abundance of putrefactive anaërobies, especially of *B. aerogenes capsulatus*, gives a peculiar character to the intestinal contents. The organisms attack carbohydrates and proteins vigorously, and butyric acid is formed from both, together at times with propionic, caproic or valeric acid. These acids are largely responsible for the odor of the stools. From proteins, besides these

acids, hydrogen, carbon dioxid and perhaps methane are formed. As a consequence the feces have a low specific gravity and often a decidedly light color. The presence of hydrogen leads to the extensive reduction of bilirubin and other pigments. "The Schmidt test with mercury bichlorid gives a strong pink color." (See page 116.) "The stools have an acid reaction, although the acids are neutralized in part by ammonia and other bases formed in the process of putrefaction. It is probable that the ammonium butyrate acts as an irritant to the gut, causing soft stools or diarrhea. Indol is absent, or present in small amounts. Phenol occasionally is found in slight excess. In the urine the ethereal sulphates at times are excessive, although the reason for this is not clear. Mercaptan may be present in the feces as a trace; it also is found in cultures by means of the isatin-sulphuric-acid test. It has been noted that as the patient improves the mercaptan becomes less or disappears, but the explanation of this phenomenon is at present unknown."

Combined Indolic and Saccharobutyric Type.—"Examples of this type of intestinal putrefaction are common. There are many putrefactive anaerobes in the gut, and also a persistent and well-marked indicanuria, which is but slightly influenced by diet. The nervous symptoms are relatively prominent and appear early: emotional irritability and periods of mental depression. Muscular or mental activity soon induces marked fatigue. Later the blood disturbances may appear. Although these patients have intervals of improvement that continue for months, on the whole the general tendency is downward. They become less robust and recuperate less promptly from every succeeding attack. They may run along for ten to fifteen years in a weak condition, with periods of slow improvement, and finally may present the picture of pernicious anemia. In others the nervous symptoms increase and the patients may need treatment in a sanitarium or in an asylum for the victims of melancholia.

"These various manifestations in different individuals may represent merely a differing reaction to the same poison. Whether the nervous system or the blood shall bear the brunt of the attack is determined by the relative vulnerability of these tissues in that particular individual. It is noticed also that under treatment one group of symptoms may improve quite independently of the other."

Symptoms. The external appearance of the patient may be the first indication of the presence of intestinal toxemia. The patient has a sickly expression, a pale yellowish complexion, and a morose disposition, forehead and cheeks are prematurely wrinkled and have brownish spots, the lips, in comparison with the pale complexion, are very hyperemic and swollen. The skin is dry and scaly, the nails soft and fissured. The lumbar glands are very painful and enlarged. At night there is a tendency to perspiration.

There are also digestive symptoms, such as anorexia, dislike of meat, and great thirst. The tongue has a brownish coat, the abdomen is distended, and sometimes the liver is enlarged, especially in children. The state of the digestive organs differs in different individuals, depending upon the presence of ptosis, catarrhs, fermentation, putrefaction, constipation, enteritis membranacea, and parasites. The intestinal flora also displays characteristic signs: there is a decrease of the aerobic and facultative anaërobic bacteria and a predominance of the strict anaerobes (*Bacillus mesentericus proteus*, *putrificus*, *putridus*), which means a flora of protein putrefaction.

The so-called gastro-intestinal crises may occur, in which the accumulated enterotoxins are suddenly excreted; these cases are characterized by salivation, periodic vomiting, and periodic diarrhea. The other organs likewise suffer from the influence of the intestinal toxins. There may be cholangitis, severe icterus, and cardiac manifestations such as angina, tachycardia, bradycardia, arrhythmia, cardialgia, neuroses, and lowering of the blood-pressure. The lungs may be involved in the form of asthma and bronchitis. Inflammation of the tonsils is of frequent occurrence. The nervous system is responsible for headache, migraine, hyperchlorhydria, and mental derangement. Anemia, even the pernicious form, is not very uncommon. Urine and feces show the signs of increased protein putrefaction in the intestine (see page 121).

Considering that intestinal toxemia may, on the one hand, be the consequence of increased protein putrefaction in the intestine, and on the other the consequence of insufficiency of the antitoxic action of the various defensive organs on the normal decomposition processes, it follows that the therapy must be twofold, with the object of decreasing the intestinal protein putrefaction to normal or below, and increasing the function of the antitoxic and excretory organs.

Treatment. The intestinal culture ground on which the bacteria of protein putrefaction thrive should be changed. This is accomplished:

1. By an antiseptic diet.
2. By introducing antagonistic bacteria into the canal.
3. By antiseptic medication.

Antiseptic Diet.—In order to change the culture ground of the noxious bacteria in the intestine, it is necessary to restrict or exclude among the natural foodstuffs those which favor the development of the putrefactive bacteria, and to prescribe an abundance of those which counteract putrefaction. The foods favoring putrefaction are those that contain protein: meat, fish, eggs, and the flour of lentils, peas and beans. Meat especially increases intestinal putrefaction; the less fresh the meat, the stronger the decomposition. Fish invite putrefaction; egg albumen is less susceptible, but the

legumes given with the food increase protein putrefaction in the intestine.

The antiseptic diet (see page 174) in intestinal toxemia should consist of farinaceous and milk dishes, since milk (see page 162) in all forms, as well as the carbohydrates (with the exception of legumes), inhibits putrefaction. Milk is an antiseptic food, owing to its high percentage of milk-sugar, which liberates lactic acid and succinic acid through the action in the small intestine of the *Bacillus coli communis* and the *Bacillus lactis aerogenes*. These acids are capable of preventing the anaërobic bacteria of putrefaction in the large intestine from decomposing the casein of milk and the protein of nitrogenous foods. But pure milk alone is often not well tolerated, and it is therefore advisable to use this article of diet in the form of salicylic milk (see page 176), or as milk soup thickened with flour or other material. The same precaution should be taken with skim-milk (milk from which the cream has been removed).

A much greater effect on putrefaction is exerted by the various products of sour milk. The following may be mentioned:

Whey (the clear, transparent liquid residue expressed from milk curd coagulated with rennet or pepsin) is much used as a hygienic beverage and a dietetic remedy. Indeed, special establishments have been erected for "whey cures" in Baden-Baden, Creutznach, Leivico, Meran, and Weisbaden. In the beginning of the treatment whey is sometimes difficult to digest, but the intestine soon becomes accustomed to it. It may first be taken mixed with mineral water, but later undiluted, gradually increasing the daily quantity. It should preferably be taken on an empty stomach. Whey can also be used to advantage in the preparation of soups (see page 164).

Buttermilk, owing to its small protein and fat content and its high percentage of milk-sugar and lactic acid, is well suited to the treatment of intestinal toxemia.

Sour milk is much better tolerated than fresh milk, because it does not coagulate in the stomach and thus interfere with digestion. It slightly stimulates peristalsis and diuresis. Fresh cheese, made from either milk or cream, is recommended. Koumiss and kefir (see page 164) are products of the alcoholic fermentation of milk and are beneficial.

Aside from milk, *carbohydrates* are recognized as the best antiseptic foodstuffs. Among these the best results are obtained with the various kinds of flour and the baked foods made from them, because, owing to their tardy absorption, they reach the lower parts of the intestine, where they gradually liberate their antiseptic lactic and succinic acids. For this reason it is wise to ingest with every protein meal a large quantity of farinaceous food.

In intestinal toxemia protein foodstuffs should be restricted or

entirely excluded. The best article among them is eggs. As to fats, fresh fat which comes with the meat should be avoided, while fresh butter is allowed. Farinaceous food and milk products are to be given in large quantities. Thorough mastication is, of course, absolutely necessary. No beverages should be taken with the meals. It is advisable to arrange the daily meals so that food and drink are taken alternately and not simultaneously. After every meal an hour's rest should be taken in the dorsal or right decubital position, without sleeping.

In regard to proteins in particular, care should be taken to avoid those that constitute culture grounds for the protein bacteria. These are: bouillon, fatty soups, roast meat gravy, meat jelly, meat extract, tainted meat, and any meat which is easily decomposable (venison, raw meat, and especially fish). In serious cases of intestinal toxemia meats should be absolutely forbidden, while in all cases those that contain much purin should be considerably restricted. The same is true of the legumes.

In regard to farinaceous food: raw or cooked fruit and vegetables may be taken if carefully masticated, provided there is no enteritis; while in the presence of considerable intestinal irritation (enteritis, spastic constipation) these coarser articles of diet should be entirely forbidden. The antiseptic effect of whortleberries is entitled to special mention.

Antagonistic Bacteria.—The bacteria causing intestinal putrefaction can be attacked not only by dietary measures, but also in a direct way by introducing antagonistic bacteria into the intestine. For this purpose the lactic-acid-forming bacteria or the oriental Bulgarian bacillus are available. The proteolytic bacteria may produce their harmful effects in either the small or the large intestine; in the former case the introduction of the common lactic acid bacteria may reasonably be expected to be of benefit, since they tend to localize themselves in the small intestine. If, however, the proteolytic process originates in the large intestine, the Bulgarian lactic acid bacilli are indicated.

While the primary object of introducing lactic acid bacilli is to inhibit the objectionable activity of proteolytic organisms, it is possible that, in addition to the formation of lactic acid, other products associated with their development may be formed which also act beneficially.

The best studied and the best known of oriental curdled milks is the Bulgarian yoghurt (see page 164). This yoghurt is used as a food in Turkey in Europe and Asia Minor, in Greece and Montenegro, in Servia, Roumania, and Bulgaria. The yoghurt is prepared with a certain ferment called *maya*, which possesses special properties. This *maya* has been transmitted and preserved from time immemorial by successive cultures. Each day the fresh milk is inoculated with part of the preceding day's

yoghurt, thus perpetuating the pure culture. Yoghurt is milk concentrated to half its original quantity, the composition of which, according to Olaf Jensen, is:

Casein	7.10 per cent.
Fats	7.20 per cent.
Lactose	8.30 to 9.4 per cent.
Lactic acid	0.80 per cent.
Alcohol	0.02 per cent.

Yoghurt is said to have a good effect in intestinal toxemia. It does not act well in that form of the affection which is associated with enteritis membranacea, but in catarrh of the small intestine it renders very good service. It should be taken either with the intermediate meals (10 A.M. and 4 P.M.), or with the principal meals with farinaceous dishes, rice, pudding, or as a dessert. Yoghurt may be taken for months without injury. The effect of the pure *maya* ferments and lactobacilli seems to be weaker.

Bacillus acidophilus is a non-motile, non-pathogenic bacillus found in the feces of breast-fed children and in human milk. This organism is strongly antiputrefactive and is suitable for intestinal colonization. It has been implanted within the intestinal tract instead of the *Bacillus bulgaricus*. *Bacillus acidophilus* has a greater therapeutic value because it is a normal inhabitant of the large intestine and therefore multiplies with greater rapidity.

The addition of lactose and dextrin to a protein-free diet causes a marked development of the *Bacillus acidophilus* in the intestine.

The putrefactive bacteria can also be attacked by the introduction of various kinds of yeast into the intestine. To this end beer yeast and wine yeast are utilized. Beer yeast is best administered by mouth with any kind of liquid, such as sweetened water, alkaline mineral water, or beer. Children are given one-half teaspoonful two or three times a day, before meals; adults one teaspoonful three times daily. The antiseptic effect of beer yeast is only transitory, and so is that of wine yeast. The dose of the latter for adults is one tablespoonful four to six times daily, mixed with water and a half-lump of sugar. The introduction of a pure culture of yeast in sufficient quantities causes a rapid diminution of intestinal putrefaction, but this effect is transitory.

Antiseptic Medication. The putrefactive bacteria of the intestine may further be attacked by antiseptic medication. There is no antiseptic strong enough, in doses which would be safe, to destroy the viability of the bacteria in a quantity of fluid equal to that contained in the bowel. For such an effect Horatio C. Wood states that it would require about 30 Gm. (1 ounce) of phenol or 0.3 Gm. (5 grains) of corrosive sublimate. On the other hand, it is theoretically possible to bring an antiseptic influence to bear in the intestine—that is, to restrain the development of bacteria. For this purpose, in a quantity of culture medium equal to the contents of the intestine, it would require: phenol, 12 Gm. (3 drams);

erosote, 2 Cc. (30 minims); formaldehyde solution, 0.6 Cc. (10 minims); or betanaphthol, about 2 Gm. (30 grains). These figures do not take into consideration the possibility of absorption. A critical examination of the experimental data as to the effect of chemical agents on the intestinal flora indicates that it is possible to reduce the number of bacteria in the bowel by means of antiseptic agents. The evidence shows that the most reliable official drug is betanaphthol. It passes through the stomach undecomposed and forms free naphthol in the intestine, without irritation. Betanaphthol may be given in large quantities and, as it is slowly absorbed, its antiseptic action lasts for a long time. It may be given in doses of 0.3 to 0.6 Gm. (5 to 10 grains) four times daily (see page 279).

Experimental researches upon the influence of hexamethylenamin upon intestinal decomposition clearly show that the indican in the urine examined decreases with the daily administration of hexamethylenamin and finally disappears altogether. Hexamethylenamin administered in daily doses of 2 Gm. (30 grains) to a healthy individual, living on a mixed diet, inhibits ordinary bacterial intestinal putrefaction.

According to Combe, the principal intestinal antiseptics are: hydrochloric acid; menthol, 2 Gm. (30 grains) a day; bismuth salicylate, 0.6 Gm. (10 grains) three times a day; and ichthyol. The last named remedy, in the opinion of Rodari, is not sufficiently appreciated as an intestinal antiseptic. It is necessary to prescribe it in large doses. Ichthyol should be given in capsules, each containing 0.1 Gm. (2 grains). Rodari gives two such capsules every two hours. This may produce slight stomach symptoms and eructations.

Chloramine-T in doses of 0.02 to 0.04 Gm. ($\frac{1}{2}$ to $\frac{3}{4}$ grain) three times daily will frequently give good results. It is non-toxic, is well borne in the intestine, and upon slow decomposition in the alimentary tract effectually overcomes the infectious condition and deodorizes foul stools.

Calomel, resorcinol and salicylic acid may also be mentioned in this connection.

Sulphur sublimatum is an antiseptic that can be easily used in intestinal putrefaction. Summarizing the advantages of sulphur as an intestinal antiseptic, Wild says: "It is almost tasteless and is easily administered; it is insoluble in the stomach, and the greater part of it passes along the whole length of the alimentary canal; it does not interfere with the action of any of the digestive secretions; it forms active antiseptic substances in the intestine when their contents become neutral or alkaline—some of these substances are gaseous and penetrate to all parts of the intestine; it is sufficiently non-poisonous to be given in effective doses; it has valuable laxative properties which promote an early evacuation of the intestinal contents; and it is cheap."

Putrefactive bacteria can further be unfavorably influenced by the administration of purgatives (see page 282) and by intestinal irrigations (see page 220). The principal laxatives available are castor oil, calomel, and the salines. Intestinal irrigation is indicated in stasis with intestinal toxemia. Irrigations with 1-per-cent, ichthyol are efficacious. Fleiner's oil enemata (see page 223) have given me better results than any other method of treatment.

In the stimulation of the antitoxic organs the most important point is to keep the kidneys acting freely, in order to hasten elimination. This is best effected by duodenal lavage and by the introduction of physiologic salt solution by vein or rectum.

Treating the Constipation. Many cases of chronic intestinal stasis and toxemia recover when the accompanying constipation is properly treated. It is of the utmost importance to decide whether the constipation is of the atonic or the spastic type. The differentiation is not always easy. Patients suffering from spastic constipation are vagotonic. This can be easily recognized by the positive oculocardiac reflex, by Hering's phenomenon, and by the pilocarpin test (see page 390). Spastic constipation is due to constriction or spasm of a few isolated loops of the intestine, readily demonstrable by the Roentgen ray. The fluoroscope will also show the relaxing effect of a hypodermic of atropin upon the spasm. The enterospasm may be painful or not; in the former case it is due to neuropathic conditions associated with disease of the abdominal viscera or pelvic organs. (Plate XX, Fig. 2.)

The aim of the treatment of the atonic variety of constipation must be to so improve the muscular condition by dietetic measures as to finally attain regularity of defecation with a normal supply of food. The diet should be large and bulky, rich in insoluble residue, including usually an increased amount of carbohydrate, and more particularly of foods rich in cellulose. These foods increase peristalsis by stimulating the muscular coat of the intestine (see Laxative Diet, page 182).

In the treatment of the spastic variety of constipation, bulky foods are avoided, and a variety of fruits should be given because of their chemical constitution. They stimulate peristalsis, partly because of their fruit acids, and partly because they contain sugar, which tends to increase the fermentative processes in the intestine. Easily melted fats, as well as butter, oil and cream, not only have a mechanical effect, but also act chemically, stimulating peristalsis by means of the great amount of fatty acids they develop. (See Chapter XXXVII on Chronic Constipation.)

Petroleum jelly will lubricate the whole gastro-intestinal tract, thus facilitating the passage of the contents. The lubrication of the chyme in the intestine assists in its timely removal in cases of intestinal stasis. After the due administration of this jelly the feces are softened and under the microscope are found to contain

minute oil globules. Petroleum jelly of the best quality seems to act quite as well as the Russian mineral oil (see page 582); it is heavier and therefore mixes more thoroughly with the feces; at the same time its viscosity prevents it from passing through the bowel too rapidly. The jelly, when pure, is not absorbed from the alimentary tract, and even in large doses has no poisonous effect. It is useful not only as a lubricant, but also as a means of healing superficial lesions of the mucous membrane (see page 650).

Lavage.—In cases of intestinal stasis associated with an obstruction, such as kinks, bands, membranes and adhesions, I have found that duodenal lavage (see page 105) clears out the whole of the intestine above the obstruction and gives the bowel an opportunity to recover tonicity sufficient to effectually overcome the stasis. If there happen to be adhesions, compensation takes place in some way or other, and recovery is the result. The kink may remain the same, but the patient recovers his health, which after all is the practical object of any treatment in any condition. As long as normal motility is not interfered with, there is absolutely no indication for surgical intervention. Lavage restores normal physiology, and if it is true that the duodenum excretes a poison which is responsible for a great deal of the toxemia from which patients suffer, then duodenal lavage is the remedy to remove the poison. I am convinced, from the results obtained in the majority of cases of intestinal stasis, that duodenal lavage will do all that surgery can do, and do it better, since it does not entail a train of possible deformities and their consequences.

Any of the many duodenal tubes can be employed (see page 99). With a little practice the tube can be easily manipulated to ensure its arrival at the pylorus, whence it is carried by peristalsis into the duodenum, with the patient lying on his right side. The pylorus should be patulous, and it is necessary therefore to undertake the proceedings on an empty stomach. By any of several well-known tests (see page 100) we can ascertain whether the end of the tube has arrived in the duodenum; and as soon as it has, lavage may be begun.¹

For the treatment of intestinal stasis in general, the best solution is 30 Gm. of magnesium sulphate and 30 Gm. of sodium sulphate in a liter of water. The lavage is given daily for ten days, as a first series of applications; then on alternate days for another ten days; and the third series follows at intervals of three days, the number of treatments given in this last series being only three or four. Although by this time success is fairly certain, I have made it a practice to continue with one lavage a week until recovery is fully established. There are no unpleasant by-effects, and patients will tolerate the treatment for any length of time without discomfort (see page 106).

¹ Charles D. Anon. Treatment of Intestinal Stasis by Duodenal Lavage, Medical Record, August 17, 1918.

Mechanical Treatment. The mechanical treatment consists principally in the use of an abdominal bandage which will furnish a suitable support to the relaxed abdominal wall. This treatment acts beneficially by ameliorating the symptoms due to tension or stretching of the mesenteries. For details as to application see page 574.

Surgical Treatment.—Surgery is now frequently employed for the cure of intestinal toxemia associated with chronic intestinal stasis. A Roentgen-ray examination (see Chapter V) with the bismuth mixture may show a displaced stomach, a prolapsed colon, kinking of the hepatic or splenic flexure, spasms of different loops of the intestine, or the presence of bands, membranes and adhesions; but such conditions do not imply that surgery is inevitably necessary. So long as motility is not interfered with, there is no absolute indication for surgical intervention. A transverse colon can be displaced anywhere from its normal position down to the symphysis without interfering with motility. The cinematograph shows that such a displaced intestine can empty itself properly even if the angulations at the distal ileum and the hepatic and splenic flexures show absolute kinks. It has been proved that stasis is not due to an abnormal position of the intestine (kink, ptosis, or redundant colon) so long as there is no actual mechanical obstruction.

Recent experimental work by Keith¹ explains the mechanism of intestinal movements, and seems to account for the production of intestinal stasis upon a physiologic basis. In his histologic studies he discovered a nodal tissue (see page 64) intermediate between nerve and muscle and interposed between Auerbach's myenteric plexus and the smooth muscle of the intestinal wall. This intermediate tissue possesses two distinct functions—one, the initiation and regulation of the muscular contractions in the segment of the intestine which it controls; the other, the power of conducting impulses which lead to the forward propulsion of the intestinal contents. Not only do the demonstrable physiologic functions of these "nodes" explain the normal movements of the intestine, but it is obvious that a perversion of the function of any one of them is capable of giving rise to an inhibition of the forward progress of the intestinal contents, with resulting intestinal stasis. In the establishment of this as the physiologic explanation of the mechanism of the production of intestinal stasis, Keith was able to demonstrate the presence of definite fibrotic and degenerative changes in this nodal tissue in segments of the intestine extirpated for the relief of chronic intestinal stasis. From these investigations he concludes that it is improbable that mechanical conditions or derangements of sphincteric action underlie the production of intestinal stasis, but rather that the true cause is the production

¹ A New Theory of the Cause of Enterostasis. The Lancet, August 21, 1915, p. 375.

of some "block" or disorder in the nodal and conducting system of the intestine analogous to the heart block and similar disturbances of cardiac function. He does not accept Lane's "drag, band and kink" theory (see page 561).

By short-circuiting the ileal contents directly into the sigmoid, or by the extirpation of the colon, Lane has succeeded in curing coincident pyorrhea alveolaris, tuberculosis, arthritis deformans, nephritis, cystitis, pyelitis, endometritis, salpingitis, exophthalmic goiter, skin disease, colitis, endocarditis, epilepsy, neurasthenia, and a host of other diseases. An operation like that of colectomy is an extensive and dangerous one, and seems hardly justifiable in the treatment of such chronic joint diseases as arthritis deformans or the arthritis of tuberculosis. It is surprising and a bit confusing to hear the cure of so many varied and unrelated diseases attributed to one remedial operation. The connection which is asserted between chronic intestinal stasis associated with intestinal toxemia and the many forms of ill-health which the short-circuiting operation is said to cure, is not convincing. In view of the radical treatment urged by the followers of Lane, and the confidence placed in its not yet entirely tested results, internists will do well to cultivate a sane conservatism. We are not warranted in encouraging surgeons to hazard the operation of short-circuiting and colectomy unless we have a definite organic intestinal obstruction to deal with (see page 668).

CHAPTER XL.

FLATULENCE, METEORISM, AND TYMPANITES.

DISTENTION of the stomach or intestine with air or gas is known as flatulence. When the gas accumulates to such an extent in the intestine as to cause acute symptoms, we call it meteorism. A chronic condition of gas accumulation in the intestinal canal we call tympanites. Normally the intestine always contains a certain amount of gas. A part of this gas is atmospheric air which has been swallowed with the saliva or taken with the food. In this way a great deal of oxygen and nitrogen is introduced into the stomach and intestine. Oxygen is quickly absorbed and is therefore never found in the lower intestine.

Origin of Gases.—The intestinal gases consist for the most part of hydrogen, nitrogen, carbon dioxide, methane, ammonia, sulphuretted hydrogen, with very small quantities of volatile fatty acids. The nitrogen is derived from atmospheric air introduced during the act of deglutition. The other gases are generated by the decomposition of the ingested food through the action of the intestinal bacteria. A small percentage of all articles of food (protein, fat, carbohydrate, cellulose) is broken up by bacterial activity. Carbon dioxide is derived from the fermentation of the carbohydrates, a process which yields also lactic acid and alcohol; a small portion is due to the fermentation of cellulose, which splits up into carbon dioxide, methane, hydrogen, acetic and butyric acids. The decomposition of protein produces carbon dioxide, hydrogen, and ammonia. Sulphuretted hydrogen is generated principally after certain articles of food containing sulphur have been eaten, such as onions, garlic, and horseradish.

The smallest quantity of these gases is expelled by way of the anus. The greater part is absorbed and excreted with the air during respiration. Carbon dioxide is absorbed most readily and hydrogen most tardily.

Flatulence may be defined as a condition in which the presence of gases is disagreeably apparent, both subjectively and objectively. This may occur:

1. With an abnormally large collection of gas, the evacuation of which is impeded.
2. With a normal quantity of gas, the evacuation of which is impeded.
3. With overformation of gases, and interference with their expulsion, both at the same time.

4. When the intestinal gases become objectionable, even though the quantity is normal and expulsion not impeded. These various forms of flatulence must be recognized, in order that an accurate diagnosis may be made and proper treatment instituted.

1. The formation of abnormally large quantities of gases may be brought about through either the exogenous or the endogenous route.

Exogenous Gas.—The exogenous introduction of intestinal gases takes place to a slight extent during the normal act of deglutition, but may attain excessive degree in aerophagy (see page 401) and with nervous, rapid eaters. The flatulence in these patients is characterized by the expulsion of large volumes of odorless gases (nitrogen), although the intestinal functions are perfectly normal, and neither the quantity nor the quality of the food has any appreciable influence on the formation of the gases. The feces do not putrefy or ferment when kept in an incubator.

Endogenous Gas.—The endogenous abnormal formation of gases is an alimentary one, i. e., it is brought about by decomposition of the contents of the intestine. In occasional instances all articles of food give rise to increased formation of gases, especially when the bacteria of decomposition predominate in the intestinal tract. The beginning of abnormal decomposition of various kinds of food is not infrequently in the stomach. When there is achylia or motor insufficiency, the ingested food becomes contaminated with the organisms of putrefaction and fermentation. We know that microorganisms have their habitat in the healthy as well as in the diseased body, and that the production of gas is absolutely dependent upon their presence. The function of hydrochloric acid in the stomach is to assist digestion and to destroy bacteria, but it is not a perfect sterilizer; it does not destroy all the germs. Under normal conditions free hydrochloric acid arrests fermentation, but may not altogether prevent it.

Microorganisms.—In all degrees of lack in the secretion of hydrochloric acid the food that has been taken into the stomach becomes to a certain extent a medium for the propagation of bacteria. In case abnormal fermentation follows, gas is rapidly formed. In infants the gastric glands are not fully developed, and, with fermentation of the food, fungi thrive. A similar condition exists in adults in all degrees of catarrh of the stomach. Fungi enter the stomach, multiply freely, and establish nidi for propagation. Thus the stomach may become the direct cause of the gas, having allowed the microorganisms to pass on, unchallenged, into the intestine, where their multiplication is rapid.

Bacteria which have passed through the stomach multiply rapidly in the duodenum, and continue to increase until they become extremely numerous in the large intestine. Fermentation takes

place in the stomach and upper intestine. Putrefaction always takes place in the large intestine. It is estimated that an unclean mouth can harbor over one billion bacteria. These are taken into the stomach with the food and here find a medium for rapid propagation.

Flatulence may result from food decomposition. The large percentage of blood remaining in meat explains many cases of offensive decomposition of protein—a fact to which Boas has directed our attention. Raw meat and sausage containing blood are special offenders in this respect. Eggs often give rise to the formation of large quantities of gas, especially when they are not fresh. Ordinary milk also decomposes easily, just as kefir and yoghurt do. Of the carbohydrates, the coarser varieties are particularly liable to decomposition because they contain a large amount of cellulose with the starch (see Intestinal Fermentative Dyspepsia, page 677). Pure white patent flours of various kinds are usually well digested. Foods containing large quantities of cellulose are liable to induce flatulence as a result of cellulose fermentation. Fats, instead of being normally digested, may become affected by bacteria and decompose, with the formation of carbon dioxide and hydrogen. In these alimentary cases it is often difficult to correctly determine what component of the food produces the flatulence, and it may require much time and patient study to decide the question. Meteorism is not always present in this form of flatulence.

2. Flatulence in consequence of impeded expulsion of gases, although the quantity may be only normal, occurs in chronic constipation. This is especially apparent in cases of organic obstruction; that is, in organic stricture of the intestine. In such cases flatulence is often the very first symptom; it is usually accompanied by meteorism and tympanites. In every case of chronic meteorism a careful examination should be made for intestinal stenosis. To this class belongs the flatulence which occurs in heart disease from disturbance of the circulation which supplies the abdominal organs, that due to general arteriosclerosis, and to sclerosis of the abdominal vessels. In these conditions the flatulence is a result of diminished absorption of gases by the mucous membrane of the intestine, induced by disturbances in the circulation. In all these cases the flatulence may be accompanied by colicky pains (*colika flatulenta*).

3. The disturbances mentioned under "1" and "2" may become combined, thus inducing an extreme condition of meteorism.

4. It is not uncommon for nervous patients suffering from neurasthenia, hysteria and hypochondriasis to complain of flatulence when the quantity of the gases does not exceed the normal and there is no obstruction to their escape.

Treatment. A consideration of the preceding statements leads to the treatment as detailed below. The exogenous flatulence of aerophagy and of rapid eaters is not to be treated by dietary instruc-

tions, but by systematic physical and hydrotherapeutic measures directed to the whole nervous system. As aerophagy (see page 461) is a distinctly nervous affection, it is necessary to educate the patients to a regular hygienic method of feeding. These persons must gradually learn to eat slowly, thoroughly masticate their food, and avoid talking at meal time.

For the past few years it has been generally admitted that flatulence may exist for a long time without any serious consequences. In most instances there is no actual increase in the fermentation of the food. The frequent eructation of gas, or belching, is explained on the theory that swallowing of air takes place at frequent intervals, almost unconsciously (aerophagy), and the patient instinctively endeavors to get rid of it. We should absolutely forbid this practice. Patients must be instructed to allow the gas in the stomach to enter the intestine, where its absorption and elimination are rapid.

It has been repeatedly proved that enteric pressure favors the rapid absorption of gas. Whenever the existing pressure is lessened by the frequent opening of the cardia and rectum, absorption is delayed. Habit thus plays an important part in perpetuating the very condition which it is intended to relieve. The digestive canal can be trained to master the gases by quick absorption. When the attention is actively kept up by some interesting or important subject (also during sleep) the gases do not escape from the digestive canal. It is only in easy-going hours, when one's thoughts are turned to the vegetative functions, that the need of passing gas makes itself felt. There is, however, no necessity of immediately responding to this sensation. The patient should be impressed with the fact that no harm will result from retaining the gas. He should try to keep back the belching as well as the passing of flatus whenever possible, instead of yielding to the inclination of the moment.

Diet. Conditions in alimentary flatulence are quite different. Here the diet should be regulated most carefully. The amount of food taken at one time should never be large. Care should be taken to have the food cut up minutely and well masticated, since large particles are poorly digested and prone to decompose. The physician should be careful to ascertain from the patient the particular kinds of food which have been found to disagree with him and to cause flatulence. By this means it is often possible to acquire valuable information as to what articles of food should be excluded from the diet. Many times the examination of the feces after a test-diet (see page 112) shows whether the carbohydrates or the proteins should be restricted. Very often the carbohydrates are at fault, and then we should restrict the coarse carbohydrates, as rye bread, vegetables rich in cellulose, and potatoes. The finer varieties of white flour, white flour pastry, dextrinated

flours and a pure protein-fat diet should be given for a time. In all cases of flatulence, raw and rare meat should be prohibited because of the blood it contains; meat should always be thoroughly roasted or boiled. One should be very careful with eggs, permitting the use of strictly fresh ones only. It is sometimes necessary to eliminate milk, kefir, and yoghurt, or the milk must be given with salicylic acid. One liter (quart) of milk is boiled with 0.2 Gm. (3 grains) of salicylic acid. The salicylic acid is first triturated with a little milk in a mortar, and when minutely divided is added to the total quantity. Onions, horseradish, lettuce, potatoes, cabbage, beets, peas, beans and radishes are to be forbidden in every case. It is, however, frequently difficult to find the proper diet for each individual case; great patience is necessary. The incubator test (see page 116) should be employed to determine whether the gas is due to putrefaction (of proteins) or fermentation (of carbohydrates). If the gas is due to putrefaction, an antiputrefactive diet (see page 174) is indicated; if to fermentation, an antifermentative diet (see page 181). It should be remembered that the odor of the gas in putrefaction is always fetid, while there is little or no odor to the gas of fermentation. This point is of great value in outlining the diet, especially when the incubator test is not available. Artificial and natural mineral waters containing carbon dioxide gas are to be forbidden. Should constipation be a feature of the case, it should receive proper treatment (see page 659).

Massage.—Abdominal massage (effleurage) will often be found very useful in such cases by mechanically forcing out the gases (see page 211). The use of a cannon ball (weighing two to five pounds) has been found very valuable; the ball should be rolled over the large intestine, beginning with the cecum, for five minutes every night, and the treatment continued for some little time.

Medication. An important place was formerly held by medication in the treatment of flatulence. Great virtue was ascribed to the various teas (valerian, peppermint, caraway, fennel), to the ethereal oils (oil of peppermint, oil of anise, oil of fennel), and to wood charcoal (carbo ligni). At present we are in a position to deny that these products possess any curative properties whatever. If patients praise them, their praise can refer only to the agreeable carminative sensation they produce for a moment. Particularly of wood charcoal we know that it is able to exert its antiseptic and absorbing powers only when dry. There is no medicament available that will prevent the formation of gas in the intestine. All drugs employed to prevent decomposition of the intestinal contents have thus far failed. The absorption of gases by medicine is an illusion. A diminution in the processes of decomposition in the intestine can only be expected from antiseptic agents. Of these the best is salicylic acid, particularly in the form of magnesium salicylate, 1 or 2 Gm. (15 to 30 grains) three times a day. Mag-

nesium salicylate has the valuable property of not inducing constipation. When the bowels are lax, bismuth salicylate should be given because of its astringent effect.

For lactic acid fermentation, ammonium fluorid (a substance employed as an antiferment in breweries and distilleries) in solution may be given as follows:

	Gm. or Cc.	
R—Ammonii fluoridi	0.2-0.5	gr. ii-viiss
Aquæ destillatæ	300 0	℥ x
Misce.		
Sig.—Tablespoonful after meals.		

Sulphur iodid in doses of 0.1 Gm. ($1\frac{1}{2}$ grains) in a capsule is an effective remedy for the fermentation which accompanies flatulence, with periodical eructation of gas produced in the stomach, as distinguished from that due to air-swallowing (which is almost continuous, and for which the remedy named is of no avail). Precipitated sulphur checks lactic acid fermentation and stimulates intestinal contractions; it should be given in a dose of 1 Gm. (15 grains) mixed with an equal amount of calcined magnesia, after each meal.

Physostigmin is warmly advocated for the expulsion of gases. The salicylate should be given according to the following formula:

	Gm. or Cc.	
R—Physostigminæ salicylatis	0.003-0.005	gr. $\frac{1}{20}$ - $\frac{1}{16}$
Extracti gentianæ, q. s.		
Misce et ft. pil. no. x.		
Sig.—One pill two or three times a day.		

Severe meteorism following operation is often relieved by the hypodermic injection of physostigmin salicylate in doses of 0.001 Gm. ($\frac{1}{10}$ grain). The drug is well borne, even by patients who are much weakened. The use of physostigmin is especially recommended in cases of postoperative paresis of the intestine, the so-called pseudo-ileus. The hypodermic or intravenous injection of Pituitary extract will stimulate peristalsis and eliminate flatus. It is necessary to give it in larger doses than those employed for stimulating uterine contractions in inertia uteri. A dose of 2 Cc. (30 minims) may be given, and repeated at hourly intervals until three or four doses are given if necessary. Hormonal also induces strong peristalsis and the passage of flatus (see page 667); it should be given intravenously.

In very obstinate cases chronic flatulence may be treated by means of intestinal tubes. With the patient recumbent, an intestinal tube is introduced a few inches beyond the sphincter up into the rectum, and left there for an hour, perhaps several hours. The best instruments for this purpose are stiff rectal tubes of hard rubber with a wide opening which does not become readily obstructed. (Soft tubes are liable to kink, thus becoming blocked.) In this

manner it is sometimes possible to facilitate the escape of large volumes of gas. (See Chapter XI.) Enemata, especially of turpentine, are frequently very effectual. The Noble formula is particularly valuable (see page 752). Cold water is often more likely to stimulate peristalsis than warm water.

When the accumulation of gas is due to a stricture of the intestine, it may be necessary to administer purgatives to diminish or inhibit the gas accumulation. Only the milder ones are to be employed—for instance, small doses of castor oil, or the saline cathartic waters. The diet must be regulated at the same time. (See Chapter XLVII.)

When flatulence supervenes in consequence of cardiac disease or arteriosclerosis, these etiologic diseases should receive careful attention. Systematic massage is frequently of benefit, the absorption of the gases being favored by the outflow of the venous blood induced by it.

CHAPTER XLI.

ULCERS OF THE INTESTINE.

DUODENAL ULCER—VICUS ROTUNDUM DUODENI—PEPTIC ULCER OF THE DUODENUM; JEJUNAL ULCER.

DUODENAL ULCER.

DUODENAL ulcer implies a disintegration and necrosis of tissue in the duodenum. It may consist of unhealthy granulations becoming necrotic and gangrenous. In the majority of cases the ulcer is on the anterior wall of the duodenum, within two centimeters of the pylorus. The pyloric vein is regarded by surgeons as a line of demarcation between a pyloric and a duodenal ulcer. The ulcer often appears as a simple roundish depression in the mucous membrane, having the muscular coat as its floor. In other cases a layer of thickened peritoneum forms the base; or it may rest on the pancreas, liver, or gall bladder.

Etiology.—Curling has called attention to the fact that a severe burn or scald upon the surface of the body is sometimes followed by ulcer formation in the duodenum. This has often been clinically demonstrated. Uremia may cause duodenal ulcer, particularly in the presence of nephritis. Etiologic factors may be found in a thrombosis of the vessels supplying the duodenum, tuberculosis, traumatism, toxins, and bacterial infections.

Some clinicians believe that hyperacid chyme in the duodenum may originate the ulcer by its effect on the mucous membrane. As soon as this chyme has been neutralized by the alkaline juices of the duodenum, it ceases to irritate. Owing to anatomic formation, the alkaline juices reach a higher level in females than in males, and it is a fact that there are fewer cases of duodenal ulcer in females than in males.

There is a possibility that duodenal ulcer is caused by the absorption of toxic substances from the vermiform appendix or the colon. In 66 per cent. of his cases of duodenal ulcer requiring operation Puterson found appendicular disease also. In none of these cases was there any clinical reason to assume the existence of disease of the appendix. It has been estimated that 36 per cent. of all cases of ulcer of the stomach and duodenum are associated with a diseased appendix. In 15 per cent. of the ulcer cases, cholecystitis and cholelithiasis are present. It would seem that the exciting cause is bacterial, proceeding from the appendix or the gall bladder.

La Roque believes the original source of the bacteria is some pathologic area of the abdomen drained by the portal vein, while Rosenow says the focus of infection is a diseased condition of the teeth, gums and tonsils, with its accompanying oral sepsis (see page 290).

It has been demonstrated experimentally that an emulsion of a freshly excised, macerated duodenal ulcer injected into the ear of a rabbit will frequently produce an ulcer in the stomach or upper intestine. Steinharter produced gastric ulcer in rabbits by intravenous inoculations with clumped colon bacilli, and more recently with staphylococci. It thus seems reasonable to assume the possibility of a secondary infection from a primary focus (see page 480).

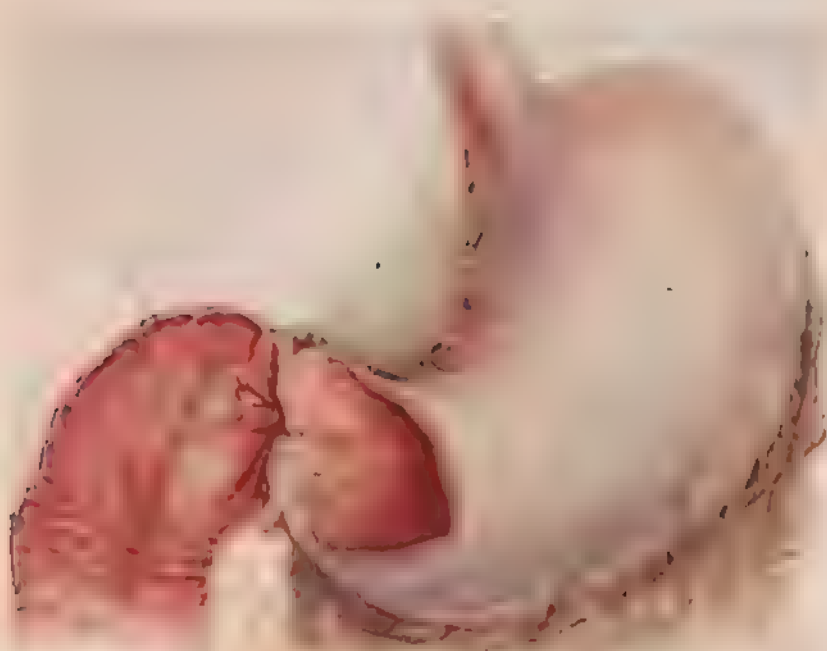
Duodenal ulcer is of more frequent occurrence than was formerly assumed by pathologic anatomists. It is a more insidious disease than gastric ulcer. Though more often associated with complications, such as severe hemorrhage and perforations, the symptoms before the onset of complications are much lighter than those of gastric ulcer. (See Plate XXVII.)

Symptoms. The anamnesis is of the greatest importance in the diagnosis of duodenal ulcer. The patient complains of a feeling of burning and fulness in the gastric region two to four hours after meals and at midnight, and describes it as a hunger pain. These symptoms promptly disappear upon ingestion of food or alkalis. The symptoms manifest themselves periodically and may have existed ten to twenty years or even longer. In typical cases the pain is located in the hypochondriac region in the right parasternal line, about two centimeters below the arch of the ribs. The patients may have symptoms of digestive disorder, of which they have complained as long as they can remember. The ulceration may begin early in life and continue, the severity of the symptoms varying upon the degree of destruction of tissue. In some cases, instead of ulcer of the stomach, the patients complain of burning pain two or more hours after meals; directly after the meal there is no pain. At times the pain may be relieved by eating, but in even the most severe cases. The patients are usually pale, and the tongue is coated with a yellowish film.

The patients may lose weight, and in some cases the weeks or months may be passed in bed. The mortality is about one per cent. The disease is more common in men than in women. The symptoms are usually periodic, and the patients are usually pale and thin. The pain is usually located in the right hypochondriac region or in the right parasternal line, about two centimeters below the arch of the ribs. The patients may have symptoms of digestive disorder, of which they have complained as long as they can remember. The ulceration may begin early in life and continue, the severity of the symptoms varying upon the degree of destruction of tissue.

The patients may lose weight, and in some cases the weeks or months may be passed in bed. The mortality is about one per cent. The disease is more common in men than in women. The symptoms are usually periodic, and the patients are usually pale and thin.

PLATE XXVII



Perforating Duodenal Ulcer with Hypertrophy of the Pylorus
and Dilatation of the Stomach.

appendicitis, or cholelithiasis. Many cases of duodenal ulcer are complicated by more or less pronounced neurasthenia exerting an influence upon the symptom-complex. There is a disturbance of the vegetative nervous system (see page 387). There are often vasomotor disturbances which manifest themselves in cold hands and feet. Some cases are complicated by psychic excitation (see page 389).

These cases often remind us of hyperacidity and hypersecretion. But pains at night in cases of pure hyperacidity or of gastric hypersecretion are certainly rare; nor does hunger pain occur very often in uncomplicated cases. In gastric ulcer the pains usually occur one or two hours after meals, and there is less tolerance for coarse food than in duodenal ulcer or uncomplicated hyperacidity. Vomiting also occurs much more frequently in gastric than in duodenal ulcer.

Diagnosis.—For diagnostic purposes it is necessary to distinguish between two principal groups: cases in which pain, hyperacidity, severe hemorrhage or perforation has occurred, and cases in which the symptoms are not prominent.

A peculiarity of the hemorrhage of duodenal ulcer is that it is intestinal. When there is much hemorrhage the feces may look like tar or soft asphalt. Sometimes they are formed and of a brownish-black color, and the patient's suspicion of hemorrhage is aroused only by noticing a margin of blood around the feces in the water after an evacuation. When the hemorrhages are considerable they may lead to fainting or collapse. In most cases of duodenal hemorrhage the patient lacks facial color, a condition due in grave cases to reduction of hemoglobin and erythrocytes. In mild cases, however, the general manifestations are slight, though occult blood is found in the feces. Oftentimes patients attend to their business, and are quite astonished when they are told about their hemorrhages. Attention is directed to the duodenum by dyspeptic manifestations, with pressure, a feeling of fulness, and pain, two hours or more after meals. The less pronounced the gastric manifestations, the greater the probability of the hemorrhage being from the duodenum. The origin of the hemorrhage, of course, locates the ulcer. Hemorrhage from cirrhosis of the liver must be excluded, for in the early stage of hepatic cirrhosis the blood may pass directly through the mucous membrane of the intestine. Both duodenal ulcer and hepatic cirrhosis are most apt to occur in middle life, in the male by preference. To make a differential diagnosis, one should palpate the liver and spleen, and remember that gastric pains occur far less often in hepatic cirrhosis than in gastric and duodenal ulcer.

It may be easy to make a diagnosis after an intestinal hemorrhage or perforation, or it may be quite difficult. Pain, rigidity, nausea and vomiting are the main symptoms. The pain is sudden,

aggravating, unbearable. It is usually in the upper part of the abdomen. Patients lie supine, with anxious faces, flexed thighs, and tense muscles. Rigidity of the abdominal muscles is an early symptom and is always present; the muscles are so hard that they are often called board-like. Vomiting is usually present, although this symptom alone cannot be depended upon. Perforation may occur without the slightest shock; then again the shock may be most pronounced. Perforation caused by a duodenal ulcer may occasionally be mistaken for a perforating appendicitis; but this is not a serious error, since both require surgical intervention. (See Plate XXVII.)

Some cases of perforated duodenal ulcer may give no previous history of ulcer or of digestive disorder. The perforations occur while the patients are at their usual occupations or during the middle of the night while they are in bed and asleep.

Severe epigastric pain may be caused by an acute coccus kidney, so-called the acute hematogenous kidney first described by Brewer. This is an acute infection of the kidney cortex with the staphylococcus or streptococcus, and the symptoms are predominantly intestinal. Before we knew anything of the existence of this disease the lithotripsy operation was generally done for gallstones or some acute abdominal condition.

Chronic cholecystitis with or without colics may also have to be considered in the differential diagnosis, as well as chronic appendicitis and certain forms of chronic constipation. Reflex neuroses or general neurasthenia may occasionally cause certain symptoms resembling those of duodenal ulcer. All these things must be taken into account in determining the differential diagnosis. However, the combination of burning, fulness, and pronounced hunger pain relieved by alkalis or food, is almost conclusive evidence of duodenal ulcer. Periodical colics, especially when there is night pain, may enable us to differentiate from cholelithiasis, pancreatic lithiasis, appendicular colic, right renal colic, gastric ulcer, and such cases of hyperacidity and hypersecretion as are associated with them. Ulcer, fissure or erosion readily causes temporary indigestion in the presence of hypersecretion see *Pylorespasm*, page 661.

The objective findings which must be established in duodenal ulcer are a narrowed pylorus. Palpation, percussion, the determination of gastric function, and the demonstration of occult blood in the feces all have a bearing in the differential diagnosis. The latter is a great assistance see pages 435-436.

The signs of duodenal ulcer are shown in the diagram showing tenderness in duodenum shown by weight. But caution is necessary in diagnosing by weight the tenderness of duodenal ulcer and that is why the following is a good test. Reagents are examined for the presence of the following part of the duodenum

is usually located at the borderline between the ninth and tenth dorsal vertebrae; it lies at the right margin of these vertebrae and extends beyond them by two or three fingers' width; rarely is it found on the left margin. Mapped upon the surface of the body, it corresponds to a position about four fingers' width above the umbilicus, between the right sternal and the parasternal line.

A characteristic pressure point does not exist in all cases; in some, rigidity (*défense musculaire*) in the upper part of the right abdominal rectus muscle points to a pathologic condition of an organ located behind it. This muscle is often hypertrophied and resistant to pressure.

Stern describes a posture assumed by the patient which is an indication of ulcer of the duodenum: The patient, trying to stretch out his epigastrium on account of the relief obtained thereby, often prefers standing to sitting; when in discomfort while resting on a chair he sits in a slanting position in such a manner that chest, abdomen and legs form a perfect incline. When experiencing discomfort while in bed, the patient tries to lie as straight as possible, often on the left but never on the right side.

The gastric secretion frequently varies; normal secretion, subacidity and hyperacidity have all been observed—hyperacidity oftener than subacidity. Hypersecretion and continuous secretion are consistent with the existence of a duodenal ulcer. Roentgen-ray examinations have shown that in uncomplicated cases of duodenal ulcer a more or less pronounced degree of hypermotility can exist. This finding is not constant nor specific for duodenal ulcer, for hypermotility is usually found in cases of achylia gastrica. The combination of hyperacidity with hypermotility deserves special consideration. Any lesion in the duodenum raises the tone and motility of the stomach.

Examination of the feces is especially important if occult blood is repeatedly found; but reliable methods must be employed, with exclusion of meat from the diet. The benzidin test for blood is preferable to the Weber test. The positive demonstration of occult hemorrhages is significant in proportion to the number of times they are found, and the importance of this finding increases with the objective demonstration of anemia without other probable cause (see page 123).

The test for occult blood in the gastric contents is a great aid in the diagnosis of duodenal ulcer when it is preceded by an oil test breakfast (see page 626). The oil induces a regurgitation of duodenal contents into the stomach, and if the case is one of duodenal ulcer blood may then be found in the stomach contents. Gastric bleeding should, of course, be excluded by an examination of the stomach contents without the oil test breakfast.

The duodenal tube (Chapter III) should be used to confirm the diagnosis. Under normal conditions the bile extracted through

the tube contains no blood or pus cells. In duodenal ulcer we frequently find small particles of coagulated blood or, with the aid of the microscope, blood and pus cells (see page 110).

Einhorn's silk-string test should never be overlooked in the diagnosis of duodenal ulcer (see page 493). In duodenal ulcer there is usually a stain of blood on the string between 60 and 70 centimeters from the knot (Figs. 83 and 84). If this test be made several times on one individual and each time a red or brown stain is found at about the same distance from the knot, a lesion of the duodenal mucosa probably exists.

The Roentgen ray is of great assistance in the diagnosis (see page 143). Hypermotility, deformity of the duodenal cap, and pylorospasm are very important points (Plate XVI, Figs. 2, 3 and 4).

Polycythemia is frequently found in duodenal ulcer and rarely found in gastric ulcer.

There is no single symptom pathognomonic of duodenal ulcer. For this reason it is necessary to make a critical survey of all the symptoms—to strike a balance, so to speak, from positive, negative and doubtful columns of evidence. Often the diagnosis cannot go beyond a mere presumption, and in these cases it is necessary to be guided by a number of general considerations.

The first is the fact that duodenal ulcer occurs more frequently in men than in women; statistics indicate that the proportion is about four to one. Seventy-one per cent. of Mayo's cases of duodenal ulcer were in males. This may be partially explained by the mode of life led by men, including the use of alcohol and nicotine and exposure to traumatism.

Duodenal ulcer is comparatively rare before puberty, the time of its most frequent occurrence being between thirty and fifty years of age. It has been observed in the new-born and is occasionally found in young marasmic infants. These ulcers are easily overlooked—are in fact found only by those who are especially looking for them. They may be acute or chronic, and their pathologic anatomy closely resembles that of gastric ulcer in the adult. Duodenal ulcer has been reported as the cause of melena and pylorospasm in infants, and as appearing without apparent cause in children in fairly good health. These ulcers may be present even when there is no macroscopic evidence of blood in the stools; and the practitioner should make an examination with the benzdian test (see page 123) for occult blood in the feces in every case of wasting infancy. Such a procedure may furnish valuable information in the elucidation of infantile atrophy and marasmus.

Complications.—The complications of duodenal ulcer are hemorrhage, perforation, peritonitis, cicatricial stenosis, contraction of the ampulla of Vater, pancreatitis, periduodenitis, carcinoma, and diseases of the biliary ducts and the gall bladder.

Prognosis.—The prognosis of duodenal ulcer is more favorable than that of gastric ulcer, because the former is much more rarely succeeded by stenosis or carcinoma. Recovery may be expected as a result of appropriate treatment. It must, however, be borne in mind that some of these cases are so obstinate as to endanger life through their complications. The complications are usually more serious in their nature than those arising from ulcer of the stomach, though fortunately they are less frequent.

In many cases of hyperacidity or hypersecretion, manifestations suggestive of duodenal ulcer do not appear until small superficial defects of the duodenal mucosa have taken place, inducing a superficial ulcer which becomes latent as soon as it heals. We should distinguish between a light (florid) or superficial ulcer and a chronic (callous) indurated one. That the majority of duodenal ulcers are of benign character is supported by the fact that neither cicatrization nor malignant changes occur here as frequently as in gastric ulcer.

This method of reasoning will also influence the treatment. In cases of gastric ulcer there is not only the ulcer itself to be considered, but also the hypersecretion which interferes with healing.

Treatment. The internal treatment of duodenal ulcer demands practically the same attention to details as in gastric ulcer. The Leube-Ziemssen, the Lenhartz or the Sippy treatment is indicated. In stubborn cases Einhorn's duodenal alimentation cure is successful (see Chapter XXV). Above all, there should be absolute rest in every form—not only long rest in bed, but in the early stage of the treatment absolute rest of the stomach also. This is indicated not only after hemorrhages, but in duodenal ulcer causing pylorospasm, because in the latter case it is clearly important that the contractions of the pyloric musculature caused by ingestion of food should be arrested for a time or restricted.

When feeding by mouth is begun, it is well to reduce the hyperacidity by giving butter, cream, yolk of eggs, sugar and milk. If the gastric secretion is good, the stomach can digest coarser food by its proteolytic function, changing it to a thin mass. The food should, however, be free from cellulose and coarse raw connective tissue. The ulcer diet should be maintained for a prolonged period.

The medicinal treatment, which is identical with that employed in the treatment of gastric ulcer, is fully described on pages 503-506.

Dietetic and medicinal treatment is to be tried at first even in rather severe cases associated with repeated hemorrhages from the bowel, since much can be done by proper dieting and the administration of alkalis to overcome hyperchlorhydria and thus favor healing of the ulcer. In acute forms of duodenal ulcer, such as those due to toxic causes, to uremia, septicemia or surgical pro-

cedures, as well as in *ulcus neonatorum*, the best procedure consists in stimulating urinary excretion, combating the infection or other cause, and enhancing the resisting powers. In subacute forms of duodenal ulcer, such as occur when the hyperchlorhydria is in its incipency or appears at intervals only, medical treatment will usually give excellent results. In distinctly chronic types of ulcer (callous), however, which are not likely to respond to medical measures, surgical intervention is indicated. Polyvalent bacterial vaccines assist in bringing about recovery in a great many cases (see page 506).

Surgical Treatment.—The surgical treatment is to be reserved for positively diagnosed cases with recurrent hemorrhages which have defied the best internal treatment; doubtful, obstinate cases that have defied medical treatment, and in which the symptoms may be caused by cholecystitis or pericholecystitis; and cases of perforation. The treatment of perforating duodenal ulcer is always surgical. The perforation usually occurs in the anterior wall of the proximal portion of the duodenum. (See Plate XXVII and page 507.)

JEJUNAL ULCER.

Peptic ulcers of the jejunum may run a latent course to perforation, or may cause severe trouble from the beginning. Some cases have been discovered only after gastroenterostomy. Prolonged internal treatment, as for duodenal ulcer, offers better prospects of cure than surgery, except in cases of perforation. In prophylaxis, alkalis and careful dieting should be the rule after gastroenterostomy, as in the case of benign stomach affections.

A return of symptoms in a patient on whom a gastroenterostomy has been performed is often indicative of a gastrojejunal ulcer at the site of operation. Half of the ulcer is on the gastric and the other half on the intestinal side. Gastrojejunal ulcers occur in about 2 per cent. of the operated cases. Roentgenographically these cases show marked deformity about the new stoma. Etiologically, in the majority of cases, retained unabsorbable suture material is found. Proximal alimentation has given good results in many of these cases (see page 509).

CHAPTER XLII.

ULCERS OF THE INTESTINE (CONTINUED).

TYPHOID ULCERS.

A NUMBER of acute infectious diseases give rise to the formation of ulcers in the intestine. Erysipelas, variola, anthrax and septicemia are all responsible for a few cases of intestinal ulceration; but the ulcers of typhoid fever and dysentery are well known and frequently occupy a prominent position in these two diseases. In army camps, in mining camps, and in great public works bringing together large numbers of men for a longer or shorter time, there is seldom the proper care of excreta, and in many instances typhoid germs are carried by flies from the latrines and privies to food, resulting in epidemics of typhoid fever. And such carriage of typhoid by flies is by no means confined to these great temporary camps. In farmhouses in small communities, and even in the badly-cared-for portions of large cities, typhoid germs are carried from excrement to food by flies; so that the proper supervision and treatment of the breeding places of the house-fly become most important elements in the prevention of typhoid. Howard rightly believes that the insect we now call the "house-fly" should in the future be termed the "typhoid fly" in order to direct public attention to the danger of allowing it to flourish unchecked.

Dietetic Treatment. This is not the place to discuss the whole field of typhoid fever therapeutics. The reader is referred to the large works on the practice of medicine for a full discussion of this disease. But here a few suggestions may be made with reference to the dietetic treatment. In the diet of patients with typhoid fever it has been the leading principle up to the present time to spare the diseased bowel to the greatest extent possible. Typhoid fever patients, so long as the temperature continued above normal, were kept on a purely liquid diet, consisting of milk, sugar, pap, an occasional allowance of casein preparations, freshly expressed meat juice, beef tea, meat jelly, thoroughly softened stale white bread, zwieback, crackers and cocoa. At the same time analeptics in the form of alcoholic drinks were administered. Only after the complete disappearance of the fever—no rise of temperature for five to eight days—was greater variation in the diet and a little more solid food permitted. These old principles, which experience

has sanctioned, are now being assailed. Warren Coleman, particularly, pleads for a fuller diet, having found that hemorrhage of the bowel and relapses were no more frequent after more solid nourishment than with a purely liquid diet. He has observed less weakness and fewer complications in the later stages of typhoid fever when the patients had been fed better. Coleman has found that, contrary to the common belief, large quantities of selected foods can be taken without disturbance of digestion. There is no evidence to indicate that the duration of the febrile period of the disease or the range of temperature is affected by diet, except that serious recrudescences are rarer in patients who are well nourished. The mortality of the disease is reduced 50 to 75 per cent. by high caloric feeding. Shaffer and other clinicians agree with this view of high caloric feeding, and it has now come about that the permissible diet in typhoid is quite varied except in cases in which such feeding is impossible on account of somnolence and grave prostration. Various combinations of milk, cream and lactose should be given in the earlier stages of the disease. Eggs may be added to make up the protein requirement. Some of the cream may be given in the form of ice cream. In certain cases, if patients have difficulty with mastication, custards, mashed potato, cocoa, apple sauce and cereals are given, although these foods are usually more suitable later in the course of the disease. Scraped and chopped meat free of connective tissue, such as veal, chicken, game, brain and sweetbread, vegetables well cooked in the purée form, such as mashed potatoes, rice and flour porridge, mashed spinach, apple sauce and tender baked foods are now permitted or even recommended.

A high caloric diet has been proved to be of greater benefit to typhoid fever patients than a low one. Food is absorbed by typhoid patients almost as completely as by healthy individuals. Loose proper feeding the emaciation formerly seen under an exclusive bland diet does not take place. Under a high caloric diet the duration of the disease is shortened. Many patients feel so well that they are able to resume their occupation soon after their discharge from hospital. The mental condition of patients who are fed on milk is such that they are able to keep their bowels clean, so that the use of cathartics is not required. In severe milk-fed patients the frequency of hemorrhages and purities are less frequent and less profuse than in those on a bland diet. The absence of nervousness and of the tendency to hemorrhage does not occur any more than in the case of those on a bland diet. The amount of food given in the early stages of the disease should be 250 calories or more in the form of milk. A combination of solid and liquid foods may be employed in the later stages of the disease. In a recent trial and at the University of Chicago, the following results were obtained:

Food	Amount	Calories
Apple sauce	1 ounce	30
Bacon	1 ounce	210
Bread	Average slice (33 Gm.)	80
Butter	1 pat. ($\frac{1}{2}$ ounce)	80
Buttermilk	1 ounce	10
Cereal (cooked)	1 heaping tablespoonful	50
Crackers	1 ounce	114
Cream (20 per cent.)	1 ounce	60
Custard (baked)	1 ounce	180
Egg	1 (2 ounces)	80
Egg, white	1	30
Egg, yolk	1	50
Ice cream	1 ounce	100
Junket	1 ounce	35
Lactose	1 tablespoonful (9 Gm.)	38
Lemonade	1 ounce with milk sugar	120
Milk, whole	1 ounce	20
Potato (whole)	1 medium	90
Potato (mashed)	1 tablespoonful	70
Rice (boiled)	1 tablespoonful	60
Sugar, cane	1 lump	16
Sugar, milk	1 tablespoonful	36
Toast	Average slice	80
Whey	1 ounce	15

Larger quantities of each article should be given at each successive feeding, to furnish a sufficient number of calories. If the patient is given a clear soup, an egg nog may be stirred in. Each egg has a caloric value of 80, and if six eggs are given during twenty-four hours there is a total of 480 calories. Feeding at two-hour intervals has been found most satisfactory. From the above list of foods a sufficient variety can be prescribed to tempt the appetite. The aim should be to give approximately 3000 calories daily.

Houghton reviews various physiologic experiments which show that during the fever process the glandular secretions of the digestive tract are more or less insufficient. The salivary secretion is less than normal, and may even become acid. This is one of the causes of the dry mouth, coated tongue, and sordes, and is an indication for the slight salivary stimulation afforded by lemonade and orange juice. The hydrochloric acid of the stomach is greatly diminished, and it is still further decreased in amount if the food does not contain sodium chlorid. The absence of sodium chlorid from the diet was a long-continued mistake during the milk-feeding period of typhoid fever treatment. The bile and the pancreatic secretion are also decreased. It has been estimated that the ability to digest food is decreased from 10 to 12 per cent. in the typhoid patient. Hence, as the absorbable toxins from protein maldigestion are more injurious than those of starch maldigestion, the deduction seems rational that too much protein should certainly not be administered.

Chittenden and others have proved that in protein indigestion an increased amount of nitrogen does not ordinarily add nutrition or strength to the system, but is represented by an increased output

of nitrogen products in the urine, the body taking up and utilizing but a small part of the nitrogen ingested. This is certainly just as true of a typhoid patient; and the nitrogen that passes off in the urine will have entered into irritating and even more or less toxic combinations before being excreted. It would therefore seem advisable to give the typhoid patient just sufficient nitrogen for his needs, as approximately estimated. As soon as convalescence is established and the patient begins to put on flesh, he will of necessity need more nitrogen—that is, more protein.

Another effect of the absorption of superfluous nitrogen compounds is irritation of the bloodvessels, even to the point of causing vasomotor constriction. This constriction of the surface vessels during fever prevents radiation and perspiration, and as a result the skin becomes dry and hot and the temperature is increased.

If the patient receives an unnecessary amount of protein and an insufficient amount of carbohydrate during the fever, the increased quantity of ammonia compounds formed may develop an acidosis of the system or a diminished alkalinity of the blood which may terminate fatally.

It has been shown that during fever the glycogen in the liver is diminished, and unless starchy foods are administered it may entirely disappear, all the glycogen appearing in the muscles. This fact probably represents a need of the cells for combustible material; again, another indication for food that will readily form glycogen. It is also a well-known fact that the part of the body that burns first in protracted fever is the fat; and the more fat the patient has, the longer probably will his muscle and other solid tissues be protected. Also, in the absence of carbohydrate food his body fat will burn to such acid products as diacetic and beta-oxybutyric acids and to acetone, with the result of causing acidosis and death. Hence it is wise to conserve this fatty tissue by protecting it, and to prevent acidosis by the administration of such foods (fats and starches) as will furnish material for the fever process, even though it is probably impossible for the patient, under the conditions, to deposit new fat. In other words, he needs more calories than he has been receiving.

While it is well known that glycogen and sugar can be metabolized from pure protein, as evidenced in true diabetes mellitus, it is certainly a disadvantage to the system to be compelled (and especially during a fever) to make its glycogen and sugar out of protein stuff. Therefore it is quite evident that starch should form a large part of the typhoid fever patient's diet, with the addition of sugar.

The ideal estimated diet of a typhoid fever patient should therefore be: a small amount of protein, just enough to represent if possible his daily nitrogen loss; as much carbohydrate as he can comfortably digest without the production of flatulence, given in

the form best suited to the individual patient; and sodium chlorid sufficient for his hydrochloric acid needs. The patient should also receive some fat; and he should have plenty of water, enough to cause him to pass at least a quart of urine in twenty-four hours, or, better, a larger amount.

Houghton has drawn up a diet table for typhoid fever, with the amounts of protein, fat, carbohydrate and calories.

Hour	Material	Weight	Approximate amount	Percentage of			Calo- ries
				Protein	Fat	Carbo- hydrate	
6 A.M.	Choice of Toast		2 thin slices.				
	Huntley and Palmers breakfast biscuit	35 Gm.	3	8.9	1.4	60.3	125
	Zwieback	24 Gm.	2 heaping teaspoonfuls sugar	100.0	100
	Cup coffee, sugar	8 Gm.	1 dessertspoonful.	2.4	17.6	4.5	35
8 A.M.	Cream	60 Gm.	2 heaping tablespoonfuls	9.3	1.6	74.0	230
	Gruel, cream of wheat	30 Gm.	Large handful	7.6	8.2	71.6	130
10 A.M.	Oyster crackers	250 Gm.	8 ounces	6.3	Neg. ¹	43.9	190
12 M.	Vegetable soup	80 Gm.	Size of orange.	1.9	1.0	20.0	65
	Baked potato	16 Gm.	1 tablespoonful	2.4	17.6	4.5	35
	" " washed and creamed	8 Gm.	Size of domino	1.0	80.8	..	60
	Butter	24 Gm.	2 heaping teaspoonfuls sugar	100.0	100
2 P.M.	Cup hot weak tea, sugar	35 Gm.	2 thin slices	8.9	1.4	60.3	125
	Toast	60 Gm.	2 tablespoonfuls	2.8	2.9	28.2	100
4 P.M.	Tapioca pudding	30 Gm.	Large handful	7.6	8.2	71.6	130
	Oyster crackers	100 Gm.	..	6.3	0.3	76.0	350
	Rice	8 Gm.	Size of domino	1.0	80.8	..	60
	Butter	24 Gm.	2 heaping teaspoonfuls	100.0	100
6 P.M.	Sugar	35 Gm.	2 thin slices	8.9	1.4	60.3	125
	Toast	8 Gm.	Size of domino	1.0	80.8	..	60
	Butter	12 Gm.	1 heaping teaspoonful	100.0	50
8 P.M.	Sugar	250 Gm.	8 ounces	6.3	Neg. ¹	43.9	190
	Vegetable soup	30 Gm.	Large handful.	7.6	8.2	71.6	130

¹ Percentage of solid material.

² Negligible. Weights of vegetables are those prior to cooking. Butter washed to remove free acid is preferred. All foods to be thoroughly cooked—four hours for vegetables other than potato.

The Preparation of Vegetable Soup.—Sixty grams each of green or canned French peas, white dry beans, potato, rice, and noodles, and 15 grams of carrots, are boiled in water at least four hours. Enough water should be added to make one liter—which is sufficient for four feedings. The whole yields 760 calories, of which 6.3 per cent. is protein, less than 0.2 per cent. fat, and 43.9 per cent. carbohydrate. When ready to use, stir up sediment and allow the patient to eat all (including noodles), with the exception of the pea and bean skins. Onions may be added for flavor if desired.

General Directions for Feeding. The patient should be fed with a spoon by the nurse.

The food should remain in the mouth as long as convenient.

Allow water between feedings, not at feedings.

Allowances or corrections are to be made for increase of nitrogen need during the first ten days and during convalescence. At the height of fever, if the patients cannot eat the full quantity, substitute isodynamic quantities of milk-sugar. A relative decrease of weight should be reflected in the caloric value of the food on the basis of 4 kilograms of loss per week of disease.

A great many physicians do not countenance this free method of feeding, and it must be granted that in many cases the old method appears to be quite satisfactory. Experience proves, however, that the more liberal feeding in cases of typhoid fever does not cause any damage, and it may be well to give more solid food a trial in selected cases.

Typhoid Carriers.—Until recently surgical drainage of the gall bladder was the only successful method of treatment in cases of gall bladder infected typhoid carriers. In this condition we can now bring about a complete recovery by the judicious use of the duodenal tube (see page 104).

CHAPTER XLIII.

ULCERS OF THE INTESTINE (CONTINUED).

ACUTE AND CHRONIC DYSENTERY.

DYSENTERY is a specific febrile disease, with inflammation and ulceration of the lower ileum and colon. It is caused by various infectious agents, of which the microorganisms mentioned below have been differentiated:

1. Epidemic dysentery. This disease is apt to make its appearance among large masses of people living under faulty hygienic conditions and on poor food, as in prisons, insane asylums, barracks, and in war. It is caused by the dysentery bacillus of Shiga-Kriese-Flexner. This bacillus resembles the typhoid bacillus somewhat, but is shorter and thicker, non-motile and without flagellæ. Epidemic dysentery occurs very frequently in Europe.

2. Endemic dysentery is caused by the *Endameba histolytica*, Schaudinn, and prevails in tropical countries (Philippines, Central America, Southern China, Egypt, Southern Italy, Balkan States). Recent work on the endamebæ by Craig shows that *Endameba tetragena* is identical with *Endameba histolytica*. The endamebæ are roundish cells, two or three times as large as a leukocyte, and possess great motility; they form pseudopodia, by the aid of which they are enabled to insinuate themselves between the epithelial cells of the intestine.

3. Reports from the Philippines show that dysentery is occasionally caused by the *Balantidium coli*. This organism has an oval body 7 to 10 micromillimeters in length, and is usually found in the fresh feces in pairs. It produces at first a mild intermittent diarrhea which gradually becomes dysenteric in character. *Bilharzia hematobia* may occasionally produce the disease. The parasites *Schistosoma japonica* and *Schistosoma hematobium* may cause dysentery when they enter the body by way of the alimentary tract in the drinking water.

4. Dysentery can also be caused by *Lamblia intestinalis*, known also as giardia. This is a parasite found in the upper part of the small intestine of the ordinary house mouse. It is also found in the rat, cat, dog, rabbit, sheep, and guinea-pig. Man is infected by eating food soiled with the excreta. The parasite attaches itself to the small intestine by means of its sucker. It is a flagellate protozoan and may be found rapidly moving in the warm stools.

In the *endemic form of dysentery* the infection takes place chiefly through the medium of the feces, due to improper sewerage. It can also be conveyed by direct transmission from man to man and by infected water.

Pathology. The same pathologic picture is presented by the fully developed ulcerative stage in all forms of acute and chronic dysentery. In the large intestine irregular large and small ulcers, with overlapping edges, become confluent and threaten to destroy large areas of the mucous membrane. The non-ulcerated mucous membrane is dark red, swollen, covered with polypoid granulations, and in a condition of severe catarrh. To this are added abscesses, deeply situated in the intestinal wall, which may perforate into the lumen of the intestine or externally into cavities formed by inflammatory adhesions, or more rarely into the free peritoneal cavity. Thus abscesses may develop outside of but in the neighborhood of the large intestine. The parts most frequently diseased are the descending colon, the sigmoid flexure, and the rectum; but it is possible for the whole colon to be affected. The development of the ulcers varies in the different forms of dysentery. Epidemic dysentery commences with a catarrh of the colon, to which is soon added a disseminated diphtheritic necrotizing inflammation of the mucous membrane due to the influence of the bacilli of dysentery, accompanied by extravasation of blood and swelling of the follicles. When the necrotic epithelium has been desquamated, ulcers are developed at the spots denuded of epithelium, until finally the above-described appearance of a fully developed dysentery is brought about. In endemic dysentery the disease commences in the submucosa. The endamebæ which have made their way through the epithelium into the submucosa cause infiltration, inflammation and granulation, which undergo purulent degeneration and form an ulcer. Only from that spot the mucous membrane disintegrates and the ulceration develops. In the interval between the beginning of the disease and the end of the grave chronic final stage come all the steps of the various forms of inflammation of the mucous membrane and of the formation of small or large ulcers which are characterized clinically as either grave or light cases.

Symptoms. Acute and chronic dysentery are differentiated as follows: The acute form usually appears suddenly, without any prodromic signs. After a short period of catarrhal phenomena (pain, tenesmus, etc.) there are frequent evacuations of the bowel, which contain mucus with mucus, blood, and pus. During the first few days the evacuations contain some fecal matter, but later they consist of mucus, blood and pus only. When they contain much blood the disease is called *red dysentery*; when they contain much mucus it is called *white dysentery*. The stools are usually passed every hour or even three or four times a day. In the course of a few days the patient

is annoyed by tenesmus and colicky abdominal pains. Soon general symptoms make their appearance, as fever, lassitude, emaciation, vomiting, anorexia and great thirst. The disease persists in this manner for seven to ten days, and when successfully treated improvement follows in about two or three weeks, and finally recovery. The routine microscopic examination of the stools for endamebæ in all cases of bowel disease in warm climates will alone clear up the diagnosis. In the majority of cases the pathogenic organism is easily and quickly found, on examining a drop of the blood-stained mucus, thinly spread out under a cover-glass on a warm slide, with an ordinary $\frac{1}{2}$ -inch lens. Intestinal disturbances simulating amebic dysentery may be due to an ameba known as *Cruigia migrans*. Emetin is very effective in the treatment of this as well as the commoner forms of amebic dysentery.

Complications.—The disease may be complicated by perforations, abscesses, and exudates into the perienteric connective tissue. A frequent complication in amebic dysentery is abscess of the liver, caused by migration of endamebæ into that organ.

It follows from the foregoing that the disease is always a grave one and that it is impossible to form a clear opinion at the outset as to its course. Recovery, however, is altogether possible under proper treatment in both the acute and chronic stages.

Prophylaxis. During epidemics of dysentery it is essential that the stools be disinfected and that the refuse water be well drained away. The supply of drinking water must be pure—boiled if necessary. The friends and nurses must be instructed to keep themselves scrupulously clean and to disinfect their hands and clothing thoroughly.

Prognosis.—With regard to prognosis, recoveries are more apt to occur in acute than in epidemic dysentery, and much more likely than in sporadic endemic dysentery. The acute cases sometimes pursue a comparatively mild course and end in early recovery.

When recovery is not absolute, amebic dysentery may continue and the symptoms of the disease never entirely disappear. In some cases the symptoms at first entirely disappear for a considerable time, even for months, but acute relapses repeatedly follow the slightest errors in diet, colds, or some unknown cause. These relapses may become more and more serious in character, so that the patient is gradually brought to a very low ebb, as regards both his physical and his mental condition; anemia sets in, and in time there supervenes a condition of severe inanition; it may, however, be years before this stage is reached. In other cases the intestinal disturbances never yield after the acute stage has been overcome, diarrhea keeping up persistently and sometimes revealing blood or pus. When a case is seen in this stage, the diarrhea having persisted for years, it often happens that the diagnosis of dysentery is not immediately made. Unless the symptoms improve, such

The administration of ipecac through the duodenal tube (Fig. 11) has been distinctly efficacious. Beck used 4 to 8 Gm. (1 to 2 drams) of powdered ipecac in warm water or suspended in acacia mucilage. Two to six doses were given, at intervals of one to three days. There was occasional nausea and vomiting. The drug does not irritate the stomach when given in this way. Similarly, wine of ipecac in doses of 30 to 150 Cc. (one to five ounces) can be administered by instillation daily. (See Chapter III.)

Much contradictory evidence has been given on the effectiveness of ipecacuanha in the treatment of dysentery. It is largely through the efforts of Major Leonard Rogers, Professor of Pathology in the Medical College of Calcutta, that ipecacuanha has been restored to favor. In his well-known work on Fevers in the Tropics he points out that the ipecacuanha treatment of amebic hepatitis prevents the occurrence of suppurative hepatitis, or tropical liver abscess—that this complication could be entirely avoided if the treatment by ipecacuanha were employed. Suppurative hepatitis, in his opinion, may well be included in the list of maladies which recent research work has largely conquered; fatal cases in the British Army in India have, it appears, been greatly reduced within the past few years.

Credit must again be given to Rogers for the first experimental results in the treatment of dysentery with emetin, the active principle of ipecacuanha. He found that on placing a piece of mucus containing numerous active endamebæ in normal saline solution with emetin hydrochlorid, the pathogenic organisms were immediately killed and materially altered in microscopic appearance by a 1:10,000 solution, while after a few minutes they were rendered inactive and apparently killed by as weak a solution as 1:100,000. He found also that this powerful alkaloid could be safely administered hypodermically in the treatment of amebic dysentery.

The subcutaneous injection of soluble salts of emetin is now known to be a specific remedy for amebic hepatitis and amebic dysentery. The hydrochlorid of emetin is used on account of its free solubility. The dose is 0.03 to 0.04 Gm. ($\frac{1}{4}$ to $\frac{3}{4}$ grain) twice daily for three consecutive days and then on alternate days until the clinical symptoms have cleared up. It is rarely necessary to give more than 0.5 Gm. (8 grains). The extraordinary rapidity with which very marked improvement follows the subcutaneous injection of 0.03-Gm. ($\frac{1}{4}$ -grain) doses of emetin hydrochlorid is of the greatest diagnostic importance. In cases of bacillary dysentery and other non-amebic affections the drug has no material effect on the progress of the disease. The effect of this new treatment furnishes a very reliable clinical differentiation between the two types of dysentery, which is of great advantage to the clinician.

Emetin hydrochlorid can be administered intravenously. The

pulse may occasionally be tense or intermittent, but in the main the treatment is safe as well as satisfactory. The dose is 0.06 Gm. (1 grain) in 1 Cc. (16 minims) of water.

The toxic effect of emetin on the endameba is due to the benzyl esters it contains. Benzyl benzoate acts as a synergist with ipecacuanha or its alkaloids and shortens the attack. It takes the place of morphin in that it slows the peristalsis and relieves the pain and tenesmus. The 20-per-cent. alcoholic solution of benzyl benzoate should be given in doses of 2 Cc. (30 minims) in a half-tumbler of water three times daily (see page 276).

Hemorrhagic gastroenteritis has been produced experimentally with emetin by a number of investigators. It may be difficult to recognize emetin diarrhea in the course of treatment, because the symptoms and the gross appearance of the stools are almost indistinguishable from those of amebic dysentery. Increase in the doses of emetin must be made with caution.

A saline cathartic or an effective dose of castor oil is advisable as a preliminary to the emetin treatment; and this should be followed up by an occasional saline throughout the treatment. Thus not only are the dead endamebæ washed out of the bowel, but the encapsulated ones are drawn out of their protected positions and attacked by the emetin. It is these encysted amebæ that account for the recurrence of the disease after treatment. To permanently eradicate the infection it is advisable to give a second course of emetin injections about two weeks after the first, and a third course about three weeks later.

Good results are reported from the internal administration of emetin-bismuth iodid in the treatment of amebic dysentery.¹ The dose is 0.2 Gm. (3 grains) in capsule every night for twelve consecutive nights. The drug is apt to produce nausea and vomiting unless these complications are guarded against. Should there be any sign of nausea, boiling water should be sipped. During the course the patient is best kept quiet in bed. For the first few nights, or until tolerance for the drug is well established, the pillows should be removed. In the event of salivation, the saliva should not be swallowed, but expectorated or removed with cotton swabs. It is said that the curative effects of the emetin-bismuth iodid treatment are usually permanent.

The combined treatment, emetin hypodermically and emetin-bismuth iodid by mouth, gives the best results. Emetin is given in doses of 0.06 Gm. (1 grain) for five days, and the emetin-bismuth iodid in salol-coated pills in doses of 0.2 Gm. (3 grains) daily for twelve days. Treatment is begun with both medicaments on the same day.

¹ The Lancet, March 31, 1917, p. 482.

Epinephrin will promptly relieve the dysenteric pains. Administered internally it relieves not only the pain but also the nausea, as well as the hiccough which is often a troublesome complication. Ordinarily the patient is given 10 to 20 drops of a 1:1000 solution in water every hour or two.

Arsphenamine can be introduced into the circulating blood in sufficient strength to kill the endamebæ without endangering the host. Intravenous injections of 0.6 Gm. (10 grains) of arsphenamine appear to yield excellent results. Winn reports 11 cases in which the endamebæ disappeared from the stools in twenty-four to seventy-two hours after arsphenamine was administered.

In oriental countries simaruba bark is frequently employed, and to it are ascribed virtues similar to those of ipecac. Its active principle is a bitter glucoside, quassin. The bark is given in the form of a decoction (1:10, one tablespoonful every two hours), or as wine of simaruba. Uzara also has a striking and prompt effect in amebic dysentery (see page 276).

During the Boer war cases of acute dysentery in South Africa were said to improve under treatment with magnesium sulphate in hourly doses and proper diet. On admission to the hospital the patient was given:

	Gm. or Cc.	
R—Olei ricini	30 0	℥j
Tincturæ opii	1 5	℥xxiv
Aquæ menthæ piperitæ	30 0	℥j
Misce.		
Sig.—At once.		

And as soon as the bowels had been thoroughly cleared:

	Gm. or Cc.	
R—Magnesii sulphatis	4 0	℥j
Acidi sulphurici diluti	1 0	℥xv
Aquæ menthæ piperitæ	4 0	℥j
Misce.		
Sig.—To be given every hour until the stools become feculent		

As the tenesmus was relieved and the evacuation of blood and mucus ceased, the sulphate of magnesium was administered correspondingly less frequently, but was always continued for about forty-eight hours after the dysenteric symptoms had ceased. The diet consisted of arrowroot, milk, soda water, and brandy or port wine.

The bark of catechu is also a favorite drug. It is administered either as a powder, 0.1 to 0.3 Gm. (2 to 5 grains) three times a day; in the form of keratinized pills of catechu, 0.2 Gm. (3 grains) eight to ten times a day; or in the compound tincture of catechu, in teaspoonful doses six to eight times a day. The usual intestinal astringents (see page 276) are also employed.

Iodoform is also given internally, either 0.05 Gm. (1 grain) iodoform with 0.03 Gm. ($\frac{1}{2}$ grain) opium, or combined with bismuth:

	Gm. or Cc.	
R—Bismuthi salicylatis	2 0	3ss
Iodoformi	0 3	gr. v
Misce et ft. pulv. no. vi.		
Sig.—One powder daily.		

In the Panama Canal Zone amebic dysentery is treated with large doses of bismuth subnitrate. The method consists of complete rest in bed, milk diet, colonic irrigation with normal saline, and bismuth by mouth. In severe cases a heaping teaspoonful of bismuth subnitrate, suspended in a tumblerful of plain or effervescent water, is given every three hours day and night, the amount being decreased only when improvement is maintained. The only alarming symptom that may occur is cyanosis, and this quickly subsides on the administration of magnesium sulphate. It is assumed that the drug does not act upon the microorganism of the disease, but upon the associated putrefactive symbiotic bacteria, the presence of which is essential for its growth. In fifteen to twenty days after the beginning of the treatment the bismuth subnitrate passes through the bowel white and unchanged. By this time the putrefactive bacteria, which usually liberate the nascent sulphur in the proteins, are destroyed, and the bismuth is not converted into the sulphid.

Salol can also be employed:

	Gm. or Cc.	
R—Salolis,		
Bismuthi subnitratis,		
Sodii bicarbonatis	ss 0'3	gr. v
Extracti opii	0 015	gr. $\frac{1}{4}$
Misce et ft. pulv. no. i.		
Sig.—One powder three or four times daily.		

Kaolin, bolus alba, or talcum may also be used (see page 279).

The local treatment per rectum of the diseased intestine is very important and is applied in the form of medicated irrigations. Alleviation of the pains and tenesmus and diminution of the blood in the stools follow irrigations with hot solutions of epinephrin; add 10 to 20 drops of the commercial 1:1000 solution of epinephrin to 1 liter (1 quart) of water at 110° to 120° F., and inject. Or peroxid of hydrogen, methylene blue, or permanganate of potassium may be added to the hot water. Enemata of 1 to 5 per cent. silvol are often of benefit. American investigators have advised cold enemata, and the application of ice-bags to the region of the large intestine, because of the sensitiveness of the endamebæ toward cold. Some authors employ in dysentery the treatment recommended by Cattani in cholera, which consists of tannic acid enteroclysis: 2 to 2½ liters (quarts) of a lukewarm 0.5-per-cent. tannin solution is introduced slowly into the large intestine two or three times daily, and kept there $\frac{1}{2}$ minutes (see page 232).

The pains and tenesmus may be relieved by suppositories of opium, cocain, epinephrin, or extract of belladonna, or by the administration by mouth of 10 to 20 drops of epinephrin 1:1000, a procedure which has been found to be safe and harmless. Good effects may be obtained by hydrotherapeutic procedures such as moist heat and warm packs to the abdomen.

During the past few years the serum treatment of epidemic dysentery has been made the subject of various experiments. Vaillard and Depter have prepared an antidy-senteric serum which is said to have a very beneficial effect, the number of stools diminishing immediately, with subsequent improvement of the general condition. They recommend their serum as the only effective therapeutic agent in the treatment of bacillary dysentery. In cases of doubtful origin, combined treatment with emetin and polyvalent antidy-senteric serum is recommended. Shiga also has prepared an immunizing serum with which he has obtained good results in cases of dysentery caused by the Shiga-Kruse bacillus; this serum antidotes the Flexner bacillus also, but to a decidedly less extent. A polyvalent immunizing serum has been prepared by Coyne and Auché; it is obtained from a single horse immunized against three distinct bacterial cultures. An injection of 60 to 80 Cc. of a multivalent antidy-senteric serum acts remarkably well in a majority of cases. Striking results are reported in chronic relapsing cases of dysentery from the use of a small dose of stock vaccine at fixed intervals. The vaccine employed is prepared from a culture of the Shiga-Kruse-Flexner bacillus, heated to 60° C. and suspended in salt solution. Stock vaccines sensitized with antidy-senteric serum have given striking results; the single dose never exceeds 100,000,000 bacteria.

Treatment of Chronic Dysentery.—Chronic dysentery is usually amebic and the principles of treatment are about the same as those which apply to acute dysentery. If a systematic cure is to be undertaken, the patient must be kept in bed for several weeks. The diet should at first be strict and sparing, but as soon as possible more free and rich in caloric value, and free from chemical and mechanical irritants. When it becomes possible to improve the strength and the general state of nutrition, a distinct advantage has been obtained. Care in the administration of milk is required. At first salicylic acid or lime-water should be added—two tablespoonfuls of lime-water to a quarter of a liter ($\frac{1}{2}$ pint) of milk (page 176). Should this be borne well, it can be given more freely. Kefir, and particularly yoghurt, may be given with advantage at times. Pure cultures of lactic acid bacilli may be employed with benefit. In some cases exclusive systematic milk cures are very useful, but they are not suitable for all cases (see page 162). Just as in the case of acute dysentery, a constipating diet (see page 172) should be continued for an indefinite period of time.

The treatment of chronic dysentery by medication is approximately identical with that of acute dysentery. The remedies mentioned in that connection must be also considered here, especially the subcutaneous injection of emetin hydrochlorid. When the use of this drug is not feasible, radix ipecacuanhæ should be given in large doses (2 Gm.—30 grains). Every few days a dose of castor oil is given in addition. Olive oil in large doses, two tablespoonfuls up to four tablespoonfuls, three times daily, is said to act well in some cases.

In chronic cases the lesions are found to be limited to the lower half of the large intestine—descending colon, sigmoid flexure, and rectum. It is very desirable to use some medication that will destroy the endameba or bacillus and at the same time heal the ulcerated surfaces. Recently, iodoform enemata have been recommended in chronic amebic dysentery. They are administered while the patient is in either the knee-chest or the left lateral posture, in the quantity of 250 Cc. ($\frac{1}{2}$ pint) of an iodoform emulsion containing 5 parts of iodoform to 1000 of mucilage of gum arabic. The enema must be given high, and an attempt should be made by massage to propel it as far up into the colon as possible. The iodoform is allowed to remain within the bowel for ten minutes, and most of it is then washed out by two separate water enemata. Poisoning is not apt to occur, but in some individuals having an idiosyncrasy toward iodoform a severe urticaria may make its appearance (see page 236).

A combination of the emetin cure with the iodoform irrigation seems to be the most effective treatment. Many permanent recoveries have been reported from this procedure. Bismuth subgallate suspended in gum arabic or oil has been recommended instead of the iodoform. In some cases tannic acid enteroclysis or 2-per-cent. solution of sodium salicylate may be useful. Quite recently colonic irrigation with petroleum has been recommended; the amount specified is one liter (quart), which is said to reach as far as or even beyond the ileocecal valve. The oil is retained for ten to twenty minutes.

Many cases, however, do not entirely yield to treatment. Such patients have to be very cautious all the time not to commit any indiscretion in either hygiene or diet; if they are careful it is possible for them to avoid relapses and lead a fairly comfortable existence. Repeated drinking cures at Carlsbad are to be highly recommended.

Surgical Treatment.—When no cure is attained, the patients becoming more and more exhausted as the weeks pass on, surgical intervention is indicated. It is impossible to say with absolute exactness how long internal treatment ought to be persevered in; but when all the approved methods of internal treatment have on careful trial proved ineffective, it is certainly proper to consider the

advisability of an operation. This consists in the establishment of an artificial anus at the cecum (cecostomy) or at the appendix (appendicostomy), with irrigation of the large intestine from the fistula.

Hale White was the first to suggest surgical treatment for chronic dysentery. He advocated an artificial anus on the right side of the abdomen to make possible the application of local treatment in

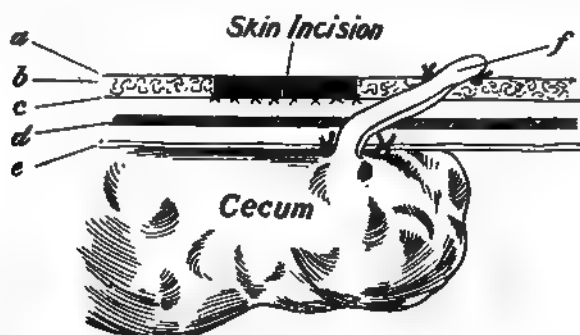


FIG. 104.—Appendicostomy: *a*, skin; *b*, subcutaneous fat; *c*, aponeurosis of external oblique, *d*, internal oblique and transversalis; *e*, peritoneum, *f*, appendix. (After Bidwell.)

intractable ulcerative colitis. Weir utilized the appendix as a means of irrigation and the introduction of medicaments into the large intestine. Willy Meyer, in 1902, applied the term "appendicostomy" to the operation devised by Weir (Fig. 104). The most striking result of this operation is the facility with which the irrigation of the large bowel can be effected (see page 735).

CHAPTER XLIV.

ULCERS OF THE INTESTINE (CONTINUED).

CATARRHAL AND FOLLICULAR ULCERS—ULCERATIVE COLITIS— ULCERATIVE ENTERITIS—ULCERATIVE SIGMOIDITIS; STERCORAL OR DECUBITAL ULCERS.

THESE ulcers were referred to when considering acute and chronic intestinal catarrh. They often develop from superficial abrasions, which are not rare in intestinal catarrh. These abrasions, being irritated chemically and infected by bacterial growths, may by further loss of substance and disintegration of tissue be converted into ulcers. Follicular ulcers may originate from inflammation of the mucous membrane or purulent degeneration and disintegration of the solitary follicles. They are principally found in the colon. Small ulcers frequently occur during the healing process of acute and chronic catarrh, and require careful treatment at this time. In severe intestinal catarrh, acute or chronic, extensive ulceration frequently makes its appearance; the small ulcers may form in such large numbers as to give an appearance of perforation to the mucous membrane, or, becoming confluent, may develop into large ulcers, the result being a most serious condition of ulcerative enteritis. Such degenerative alterations of the mucous membrane of the bowel, developed from a catarrhal affection, are found in the large intestine only. Boas designates them as cases of "colitis chronica ulcerosa," while Rosenheim's term is "colitis chronica gravis." These terms therefore indicate a disease of the large intestine characterized by the formation of numerous extensive ulcers, induced usually by acute or chronic inflammatory processes.

Bacterial invasions of various kinds probably play an etiologic rôle, since the *Bacillus coli*, staphylococci and streptococci have been found present. These ulcers are not associated with dysentery or tuberculosis. In some cases it would appear as if an achylia gastrica might have been the predisposing cause, while in others intestinal diverticula may have brought about the disease.

With respect to pathologic anatomy, it may be mentioned that these ulcers are often located in the sigmoid flexure (sigmoiditis) and in the ampulla recti, though they may be found higher up in any part of the large intestine. In dimensions the ulcers vary from the size of a pea to that of a silver dollar. In lighter cases there may be erosions instead of well-defined ulcers. There may

be areas of denuded patches or streaks over the surface of the mucosa. Here and there are scattered smaller or larger islands of discolored hyperplastic mucous membrane. In the region of these areas of denudation the mucosa is fairly smooth to granular.

Symptoms. The disease frequently commences with an acute attack, which apparently subsides within eight to fourteen days and is succeeded by a chronic stage of ulcerative colitis. This may continue for years, interrupted now and then by violent acute exacerbations of the symptoms. The first attack of an acute colitis is ushered in by fever, lassitude, headache, and pains in the limbs; the stools are diarrheic in character, containing mucus, blood and pus. The abdomen is rarely distended. The descending colon and the sigmoid flexure are painful to pressure, and resistance may be encountered on palpation. This acute condition rarely ends in complete recovery. Although the febrile symptoms subside and the stools become more solid and less frequent, the healing process is not complete; on the contrary the condition passes into a chronic stage. During this latter period the patient may feel comparatively well so that he can follow his usual employment free from all troublesome symptoms. The stool is, however, slightly more frequent than normal, while blood, mucus and pus are always present in it. Insignificant causes, as errors in diet, cold drinks, and exposure, may induce relapses and acute attacks of great frequency, gradually reducing the strength of the patient considerably. It is always very difficult to accomplish a complete cure of a well-established chronic case, but it is frequently possible to bring about improvement and periods of rest. Frequent attacks and the continuous loss of blood and secretions are apt to reduce the patient's vitality to so low an ebb as to seriously imperil life itself.

Ulcerative enteritis may be complicated by profuse hemorrhages. Perforation is not of frequent occurrence, for exudations and adhesions are usually formed around the ulcerated gut, providing an efficient barrier against it. Serous and purulent exudates often encircle the diseased intestine, especially in the left inguinal region and around the sigmoid flexure. This condition is termed *perisigmoiditis* (see Chapter I.D); it is characterized by a hard, cylindric, painful tumor in the left iliac fossa, frequently running a course similar to appendicitis with the formation of exudates and abscesses. In severe cases there occasionally results a general systemic infection accompanied by high fever, chills, swelling of the joints, endocarditis and subcutaneous hemorrhages. This morbid process is sometimes complicated by the formation of a stricture in the affected portion of the gut, due to cicatricial contraction. Taking it all in all, ulcerative enteritis is therefore a severe affection, with a rather serious prognostic aspect.

Diagnosis. Considering the foregoing, the diagnosis is not difficult. The demonstration of mucus, pus and blood in the

diarrhetic stools, with the absence of dysentery bacilli, endamebæ and tubercle bacilli, is a decisive point.

Treatment.—In the first place it is essential that the diseased bowel be placed at rest. This end is partly attained by keeping the patient continuously in bed for a long time, a procedure that must be insisted upon while the case is acute, and that is also necessary in the chronic stage of the disease in order that systematic treatment may be followed. The entire treatment is more satisfactory when carried out in an institution. Appropriate diet will assist in keeping the intestine at rest. The food should be both chemically and mechanically non-irritating, semiliquid, and at the same time as rich in caloric power as possible; it must also be distinctly antiputrefactive. Details on these points are fully considered in Chapter VII on Diet. In the chronic stage the diet need not necessarily consist entirely of soup, but may be composed of a mixture containing meat and vegetables, always finely subdivided or as purées. Caution is required with regard to milk; trials should be carefully made to ascertain whether it agrees with the patient. Yoghurt and three-day kefir are often very serviceable. Astringent foods should be given freely; the same holds good for fat, and in some cases artificial nutritive preparations are advisable. In those rare cases in which constipation prevails, fruit sauces, apple jam and honey should be added to the diet.

Opium places the intestine more completely at rest than the above diet, and it is difficult to do without it. It inhibits peristalsis, diminishes the pains, and is effective when given either by mouth or by rectum. A good substitute for opium is pantopon (see page 275). If there are pains and tenesmus, suppositories of extract of belladonna or eunydin, 0.0005 Gm. ($\frac{1}{1000}$ grain), should be given. Other remedies, which are suitable for oral administration, are subgallate of bismuth, tincture of calumba, and compound tincture of gambir. Rosenheim recommends calomel in the acute stages, giving doses of 0.03 Gm. ($\frac{1}{2}$ grain) twelve times daily for three successive days. Mercury acts especially well in some cases.

Dunn reports that in the Berkshire Asylum, Wallingford, the treatment of ulcerative colitis by creosote and oleum morrhue has given such good results that it is now exclusively utilized. He has no hesitation in saying that it relieves the symptoms more quickly and shortens the course of the disease more effectually than any other treatment, moreover, the mortality has been less since this method was adopted. The dosage is as follows: Creosote 0.1 Gm. ($\frac{1}{2}$ grains) in oleum morrhue 4 Cc. (5j), three times the first day of the disease, castor oil having been previously administered. The quantities specified are doubled on the second day and increased in the same mathematical ratio daily until on the fourth day the patient is taking creosote 0.75 Gm. (12 grains)

and oleum morrhuae 15 Cc. (3ss) three times a day. This dose is continued until the termination of the disorder.

Local treatment of the diseased bowel is appropriate and effective. It consists of a combination of lavage, colonic irrigation, and "dry treatment" (see page 237). Colonic irrigation is given in the manner described in Chapter XI. With the patient in the dorsal or left lateral position, the rectum is first cleansed with a moderate quantity of warm water, after which the water is allowed to escape. Only after this thorough cleansing should irrigations be allowed to traverse the higher sections of the diseased gut. This method prevents the possibility of inflammatory products being carried up the bowel. Carlsbad water or 0.5- to 1-per-cent. solutions of bicarbonate of sodium are used for these irrigations, and they are allowed to immediately escape. Medicated liquids, such as potassium permanganate 1:1000, boric acid 1:100, ichthyol 1:100, hydrogen peroxid (3 per cent.), solutions of gelatin, suspensions of bismuth subnitrate or bismuth subgallate in water or mucilaginous vehicles, are allowed to flow into the rectum and retained if possible for ten to fifteen minutes. These irrigations are to be made daily. Their effect is ascertained by means of the proctoscope and by microscopic examination of the discharges.



FIG. 105. Wales bougie.

Matthews' treatment is as follows: A Wales bougie (Fig. 105) is passed into the sigmoid flexure, and at least a half-gallon of tepid water containing one ounce of a saturated solution of boric acid injected. The patient retains this for twenty to thirty minutes and is then allowed to pass it. This injection is repeated each morning after the bowels have moved—for a week. Then an antiseptic astringent wash is used—one tablespoonful of *pinus canadensis* to a pint of tepid water, thrown into the sigmoid flexure daily, and allowed to remain until the patient is forced to evacuate it, repeat for six to eight days. After the second week an oil preparation consisting of sweet almond oil 1 pint, iodoform 1 dram, subnitrate of bismuth $\frac{1}{2}$ ounce, is most serviceable. This preparation should be shaken each time; one ounce of it to a teaspoonful of warm water is deposited in the sigmoid flexure, through the Wales bougie, each night at bedtime.

In case this treatment, which must be persisted in for weeks and months, and then repeated, does not accomplish the desired result, it will be advisable to give the "dry treatment" a trial. This treatment has been advanced by Rosenberg (see page 237). In order to secure satisfactory results it is absolutely necessary to

ascertain exactly how far the disease process has spread. Success can seldom be attained unless it is possible to reach far beyond the upper boundary of the disease and to subject the affected area to treatment from above downward. The opposite route must never be taken. The "dry treatment" consists primarily in the application of powder to the diseased mucous membrane, in order to permit the medication to act directly for a longer space of time than would otherwise be possible. It is, of course, necessary that the mucous membrane be previously thoroughly cleansed; and this is accomplished most satisfactorily by an enema of bicarbonate of sodium, 0.5 to 1 per cent. in water. Six to eight hours later an attempt should be made to reach the upper boundary of the inflammation with the sigmoidoscope (Fig. 46), and powder should be applied with the powder-blower (Figs. 48 and 49) until all of the mucous membrane visible to the eye is thoroughly covered. The sigmoidoscope is then withdrawn a few inches, more powder applied to the new section of mucous membrane disclosed, and so on until the anus is reached. Areas which are either too moist or covered with mucus should be first thoroughly mopped and dried with absorbent cotton. The ulcers may be cleansed with a swab saturated with peroxid of hydrogen; the generated froth forming a layer is carefully wiped away, and the powder is then applied. The ulcers may also be cauterized with nitrate-of-silver solution; the excess must be neutralized with a solution of common salt. Rosenberg recommends the following mixture for dusting purposes:

	Gm. or Gs.	
R. Zinc tannate	15-30 0	3ss-i
Magnesia usta	100 0	3ij
Mace.		
℞ Apply as dusting powder		

To which may be added, when desirable, zincform, bismuth subnitrate or zinc oxide. The application of zincform and subnitrate of bismuth, equal parts, is often followed by excellent results in ulcerative disease. The following mixture may be used for dusting:

	Gm. or Gs.	
R. Zinc formate	30 0	3ij
Bism. subnit.	30 0	3ij
Mace.		
℞ Apply as dusting powder		

The various ulcers of the large intestine should be treated by the same method as the ulcers of the small intestine, the powder being carried as far as possible.

Calomel adheres well to the intestinal mucosa and cannot be easily dislodged. It can be dusted on the mucous membrane with the powder-blower (Figs. 48 and 49) through the proctoscope. It is a non-irritant antiseptic and may be applied to the sensitive mucosa without inducing pain. Systemic disturbances do not occur even if it is used daily.

Ulcerative colitis is the very type of disease which should yield to vaccine treatment. The site of infection is localized, there is no general infection, and the symptoms are mainly local, though partly toxemic. The necessity for bacterial vaccine treatment holds good whether the primary organism concerned should ultimately prove to belong to the dysenteric, the paratyphoid, the coli or the pyogenic group. Best results are obtained after the history of the disease is understood and the germ is isolated so that an autogenous vaccine can be made. Many cases have been examined bacteriologically by Sir Almroth E. Wright, and only the streptococci and the *Bacillus coli* were found. It would seem from this that a polyvalent stock vaccine consisting of streptococci and colon bacilli could be tried with the other methods of treatment. The injection of a polyvalent vaccine, 30,000,000 streptococci and 50,000,000 colon bacilli, once a week, is a safe procedure (see page 506).

Surgical Treatment. Should none of these measures prove satisfactory, the ulcers persisting in spite of everything, and the patients becoming emaciated and severely anemic, the operative method remains, by which many cases have been successfully treated.

Active surgical treatment is necessary in cases that do not respond to internal treatment. The large bowel must be kept empty and at rest. A good-sized artificial opening is made in the cecum, or the appendix is fixed to the abdominal wall and opened (Fig. 104). Intestinal contents are in this way diverted from the ulcerated colon, which is at the same time put at rest and under the best conditions for healing. Through the opening thus made the inflamed and ulcerated bowel can be daily irrigated. The cecostomy or appendicostomy opening is easily closed.

The chief benefit secured by this operation is that the large intestine is placed at absolute rest. It is clear that the principal cause of the frequent relapses is the constant irritation of the mucous membrane by the steady passage over it of decomposed intestinal contents, so that it never has any rest. The establishment of an artificial anus through which the stools are passed without reaching the large intestine, changing the course of the fecal current, is a most urgent indication. The establishment of the *anus preternaturalis* either at the ileum or at the cecum is decidedly the most appropriate operation (see page 729).

STERCORAL OR DECUBITAL ULCERS.

These ulcers appear at those spots which are most exposed to the pressure of the fecal mass, that is to say the cecum, the splenic and sigmoid flexures, and the rectum. They do not occur when the bowel movements are quite normal, but only when the fecal matter stagnates, as in constipation and stenosis, when they may become extensive and deep, penetrating the mucous membrane. Occasionally an inflammation of the cecum, typhlitis stercoralis, develops from such ulcers. Stercoral ulcers are not very frequent. In rare cases they are followed by strictures of the gut in consequence of cicatricial contracture during the healing process.

CHAPTER XLV.

ULCERS OF THE INTESTINE (CONTINUED).

TUBERCULOSIS; SYPHILIS; EMBOLUS; THROMBUS.

TUBERCULAR INTESTINAL ULCERS.

THIS form of intestinal ulceration is more frequent than any other. Distinction is made between primary and secondary tuberculosis of the intestine. The occurrence of primary intestinal tuberculosis, running its course in patients otherwise free from tuberculosis, is now considered well established. It occurs very rarely in adults, more frequently in infants and in very young children. It is due to the ingestion of food containing tubercle bacilli, as infected milk, or meat from tuberculous animals. Secondary intestinal tuberculosis, on the other hand, is an exceedingly common disease and is found in approximately 50 to 60 per cent. of all patients affected with tuberculosis of the lungs. Its development must be ascribed to the swallowing of sputum containing the tubercle bacilli.

The bacillus of tuberculosis is not appreciably modified by the action of the gastric juice, either in form or staining reactions; the greater part of the elements which constitute the bacterial cell are not susceptible of digestion by the gastric juice, toward which the cells act in the same manner as cellulose and nuclein. This latter body, indeed, would appear to enter largely into the composition of the bacterial cell. In laboratory experiments the tubercle bacillus has retained its vitality or its virulence for thirty-six hours in contact with the gastric juice. Normal gastric juice, therefore, does not destroy the tubercle bacilli.

The favorite location of these ulcers is the lower part of the ileum and the cecum; they are also frequently found in the jejunum and in the entire length of the large intestine down to the rectum. They have their starting point in the miliary tubercles which are found in the solitary follicles and in Peyer's patches. The ulcers are of all sizes, and may be superficial or deep, penetrating to or even through the serous coat. Frequently whole regions of the mucous membrane are uniformly ulcerated; this is particularly true of the cecum. The mucous membrane contiguous to the ulcers is often in a condition of chronic catarrh of varying intensity; occasionally, however, it is perfectly healthy between the ulcers, even though there may be many in the immediate vicinity. In

severe cases of phthisis the mucous membrane of the intestine shows simultaneously extensive amyloid degeneration. The ulcers may manifest themselves subjectively by pain, which of itself does not have any special characteristics. Objectively pain on pressure is occasionally found, particularly in the umbilical region, and the abdomen may be slightly distended.

Diagnosis.—The diagnosis is made principally from the condition of the stools. Frequent attacks of diarrhea are often the first symptom of intestinal tuberculosis. These are particularly liable to occur coincidentally with catarrh of the mucous membrane of the small intestine. The diarrhea is probably due to an irritation of the nerve fibrils exposed by the deeply penetrating ulceration. The test-diet stool presents most varied appearances, depending upon the condition of the digestive tract. Aside from its diarrheic character, there are found in the feces the signs of disturbance of digestion in the small intestine, food remnants, mucus, bilirubin, decomposition products, and with concurrent catarrh of the colon correspondingly large flakes of mucus. Blood will be found in the form of occult hemorrhages, bloody mucus, or pure blood mixed with the fecal matter and visible to the naked eye. The presence of pus in the feces is of great importance; the pus can be seen with the unaided eye when the fecal matter is inspected after it has been finely triturated and placed on a black background; it appears in the form of small points the size of a pinhead, rounded and whitish yellow in color (see Chapter IV). In consequence of the admixture of blood, pus and serum, these stools always decompose. Under certain circumstances it is possible to decide whether the large intestine alone is affected, or whether the small intestine participates in the morbid process. Occasionally there are cases in which firmly formed stools are passed, or in which even constipation prevails, notwithstanding the existence of numerous ulcers. When making a more exact analysis of these cases, however, with particular regard to the feces, the presence of inflammatory products of the intestinal walls will not fail to be demonstrable. On the other hand, there may be the most marked diarrhea although only a few or quite superficial ulcers are present. The demonstration of tubercle bacilli in the feces, especially in the mucus and the little lumps of pus, is important; but it affords proof of the existence of primary tuberculosis of the intestine only when the swallowing of sputa containing tubercle bacilli can be positively excluded. The von Pirquet or the Calmette reaction will often assist in the diagnosis. The complications which may occur are: perforations, local and general peritonitis, and more rarely severe hemorrhage.

Prognosis.—The prognosis of intestinal tuberculosis is always bad. Some few ulcers may occasionally heal. Any case of tuber-

culosis, however, that has become fairly established never recovers, and a fatal termination occurs sooner or later.

Treatment.—The treatment of intestinal tuberculosis is therefore difficult and disappointing. When diarrhea exists, attempts should be made to put the intestine at rest—first, by rest in bed; and second, by dietetic measures. The diet must be regulated most carefully, especially when it can be proved from an examination of the stools that there is catarrh of the small intestine. In such cases the diet furnished should be absolutely bland, non-irritating, and not liable to decomposition. When milk can be tolerated it should always be given, with the addition of salicylic acid. Kefir and yoghurt are likewise advisable. Adolf Schmidt advises for cases in which (as ascertained by an examination of the stools) the symptoms of intestinal catarrh are receding, in which the small intestine is not involved, in which normal stools are evacuated, or in which constipation exists, that it is not necessary to be over-careful in respect to the diet, but that the dietary prescriptions should be frequently altered and a somewhat coarse diet allowed. Experience proves that a comparatively coarse diet is often well borne while a bland diet is followed by diarrhea. In such cases, therefore, the motto must be: Experiment and individualize.

Hyperalimentation (see page 569) is the *dietary treatment* of tuberculosis. Food representing the greatest caloric value should be given. The dietetic treatment should be carefully carried out, as described in Chapter VII.

Medicinal treatment may also be employed. Preparations suitable for this purpose are: Milk-somatose, the calcium preparations either alone or in combination with bismuth subnitrate, bismuth betanaphthol, and bismuth subgallate. Ulcers of the large intestine may in some instances be treated locally by medicated irrigations and enemata of bismuth subnitrate and bismuth subgallate suspensions. Here, however, great caution is necessary, as the distention of the diseased gut by these enemata may be alarming. An oil enema is prepared by thoroughly rubbing up bismuth subnitrate in a mortar with lukewarm olive oil. Occasionally we may succeed in ameliorating or even healing tuberculous ulcers of the rectum and colon by bismuth-oil enemata.

In tenesmus and pains, opium and extract of belladonna may be administered in the form of suppositories. Intestinal astringents (see Chapter XIV) such as tannoform, tannalbin, tannyl, tannigen, and tannopin are also advocated. In the presence of decomposition, enteric-coated pills of menthol or creosote can be given. Constipation is to be relieved by oil enemata (see page 223).

Symptomatic treatment of the intestinal derangements requires careful consideration, and will oftentimes of itself accomplish a great deal. Moist or dry heat should be freely applied to the abdomen for the relief of pains and diarrhea.

Concerning the *specific treatment* of intestinal tuberculosis by tuberculin, Hemmeter says there is no doubt that a number of patients have been cured by this means, cautiously employed. He has personally studied the healing of a tuberculous rectal ulcer under the influence of tuberculin; nevertheless he does not recommend the systematic employment of tuberculin in the treatment of tuberculous intestinal ulcers, enteritis, or cecal tumor, because this form of treatment has not yet been satisfactorily tested for intestinal diseases, and also because of the undesirable effects tuberculin occasionally produces on other organs which demand consideration. Good results with tuberculin imply an early institution of the treatment and the use of very small doses.

TUBERCULOSIS OF THE CECUM.

The tuberculous tumor of the cecum develops at the site of tuberculous ulcers of the cecum. The ulcers induce inflammatory infiltration and thickening of the parietes of the cecum, with pericecal inflammation and the formation of adhesions. They may also lead to cicatricial contraction, which may gradually produce a progressive stenosis of the intestinal canal and of the ileocecal valve. The stenosis favors the development of hypertrophy of the intestinal wall. Thus a tumor is being gradually developed which may assume large proportions and which is most intimately adherent to the adjoining structures by inflammatory exudations. The development of such tumors, particularly in the cecum, is favored by the fact that tubercular material may easily accumulate there. Both sexes are affected in about the same ratio. Tuberculosis of the cecum is most frequent between the ages of twenty and forty.

Symptoms.—The commencement of the disease is usually insidious. At first diarrhea alternates with conditions of constipation. After that follow most gradually symptoms of stenosis, colicky pains, visible peristalsis, and vomiting. Finally nutrition is greatly interfered with, resulting in fever and cachexia. Objectively the disease is recognized by the presence of the tumor in the cecal region, differing from carcinoma in the same locality by being more elongated. The differential diagnosis between tuberculosis and carcinoma of the cecum is frequently very difficult.

The appearance of blood, pus and tubercle bacilli in the stools is of great importance. The diagnosis is also aided by the fact that most of the patients show signs of incipient phthisis of the lungs. The diazo-reaction is usually positive in intestinal tuberculosis. The bowel may become completely occluded as the disease progresses. Other complications are: abscesses in and around the tumor, which may perforate to the outside (spontaneous formation of an artificial anus), or rupture into the peritoneal

cavity. The course of the disease may be quite protracted. Cases of two or three years' duration have been observed.

Appendicitis with induration may be mistaken for tuberculosis of the cecum. On the other hand, tuberculosis of the cecum may be complicated by appendicitis (see page 767).

Treatment.—The only treatment that offers any chance for recovery is surgical. The results after operation are not at all bad, although the patients usually continue to suffer for a long time and they generally succumb sooner or later to their lung infection. The mortality after operation is about the same, whether an entero-anastomosis with exclusion of the diseased gut or a total extirpation of the tumor has been done. The dangers of the operation *per se* are not excessive, the mortality amounting to about 16 per cent.

SYPHILITIC ULCERS OF THE INTESTINE.

Syphilis of the intestine presents itself in the form of ulcers which make their appearance quite rarely in the small and more rarely still in the large intestine. Intestinal syphilis occurs most frequently in the rectum (see page 843, "Ulcers of the Rectum"). The ulcers develop from the disintegration of gummata which are situated in the mucosa and submucosa. At first flat, bead-shaped gummatus neoplasms make their appearance, and as these disintegrate an ulcer results. The ulcers are sharply circumscribed, having a yellowish, fatty base, and extend more on the surface than in depth. Stenoses often result after cicatrization.

Treatment.—When it has been possible to establish a diagnosis, the treatment must, of course, be specific. But generally it is only possible to make a diagnosis during an operation which has become imperative in consequence of the stenotic symptoms (see page 533).

EMBOLIC AND THROMBOTIC ULCERS.

These ulcers owe their existence to embolisms in some small branches of the mesenteric artery due to an endocarditis, and to atheromatous changes in the larger arteries. A small hemorrhagic infarct develops after the embolism, and this becomes necrotic, causing an ulcer. This variety of ulcer is met with most frequently in the small intestine, and is rare in the colon. The size of the ulcers is variable and depends on the hemorrhagic infarct. In severe cases with extensive infarcts the necrotic areas may become large and the ulcerations so deep as to perforate into the peritoneal cavity. When the embolus is septic, small abscesses may develop in the submucosa, which again give rise to ulcerations. Thrombotic abscesses develop in the same manner when thromboses are formed in the inferior mesenteric vein.

CHAPTER XLVI.

OBSTRUCTION OF THE INTESTINE.

ILEUS—INTESTINAL OCCLUSION —MISERERE —PASSIO ILLACO.

IN obstruction of the bowels the intestinal canal is entirely blocked and the normal passage of the feces is completely arrested. Ileus may appear suddenly, or it may develop slowly, according to the nature of the obstruction, until complete closure results.

Occlusion of the intestine is always a very serious disease and terminates invariably in death unless the canal is reopened, either spontaneously, surgically, or by internal medication.

Etiology.—The etiology of ileus is very extensive. The following causes have been observed:

I. *External Ileus.*—(1) Angulations, kinks, membranes, occlusions due to peritoneal adhesions, omental bands and cascating glands, incarceration into preëxisting peritoneal apertures and clefts, as Meckel's diverticulum, or external and internal hernias (of special importance is hernia diaphragmatica). Meckel's diverticulum, usually from three to ten centimeters long, with its extremity free in the abdominal cavity, resembles a finger in size and shape; it is always found singly, above the ileocecal valve, opposite the insertion of the mesentery, being a remnant of the embryonal omphalomesenteric duct. These forms of ileus, because of anatomic situation, usually affect the ileum. Incarceration of a retroperitoneal hernia may cause ileus. A Trietz hernia must not be overlooked; this consists of a tumor of gas involving a part or the whole of the small intestine, although the latter may be adherent to the stomach and colon. Of diagnostic importance is the commencement of the disease during perfect health, with the sudden development of grave symptoms, complete retention of gases, and slight meteorism.

(2) Ileus in consequence of torsion (volvulus). The torsion of the intestine around its mesenteric axis affects the sigmoid flexure in two-thirds of all the cases. Volvulus of the ascending colon and of the small intestine is more rare, and that of the cecum and transverse colon most rare. Volvulus of the sigmoid flexure occurs oftenest between the ages of forty and sixty. Men are more likely to be affected than women, and the distortion occurs more frequently in connection with chronic constipation than otherwise. The course is less violent than that of ileus of the small intestine. Local meteorism is often present.

(3) Ileus in consequence of invagination or intussusception. By this is meant the invagination of one section of the gut into another section, so that finally three tubes are pushed one into another. The intussusception nearly always takes place in a downward direction and throughout a rather long distance. The intussusception of the small into the large intestine is most frequent (*invaginatio ileocecalis*); then follows the intussusception of small intestine into small intestine (*invaginatio enterica*); and finally the intussusception of large intestine into large intestine (*invaginatio colica*). The appendix vermiformis may occasionally be involved in an intussusception.

About one-half of all the cases of acute intussusception occur in children from the first to the tenth year; the more chronic variety is found most often in patients between thirty and forty years of age. The male sex is predisposed. The occurrence of the intussusception is explained either by a spasm of the intestine or by paralysis of a few single intestinal coils. The sudden onset of the most agonizing pain, later becoming intermittent, is the most characteristic sign of intussusception. Vomiting occurs very soon, but is not present as frequently as in other forms of ileus. The appearance of bloody, mucosanguineous and mucopurulent evacuations and the expulsion of gangrenous shreds of tissue, accompanied by marked tenesmus, is typical. Meteorism is seldom a prominent feature. Of great diagnostic importance is the tumor produced by the intussusception, which can be felt in many cases by palpation of the abdomen simultaneously with rigidity of the intestine during the paroxysm of pain. The tumor may, under certain circumstances, prolapse through the rectum, and most frequently does in *invaginatio colica* and *ileocecalis*.

(4) Ileus subsequent to adhesions formed between intestinal loops themselves, especially in cases of intestinal ulcers.

(5) Ileus by process of contraction in the mesentery.

(6) Ileus in consequence of compression of the gut by malignant or benign neoplasms of the other abdominal organs (appendiceal abscess, movable spleen, movable kidneys, diseases of the pancreas).

(7) Arterio-mesenteric contraction in cases of extreme acute dilatation of the stomach (see page 486).

II. *Internal Ileus*. (8) Ileus from strictures of the intestine in consequence of ulcerative processes (tuberculosis, dysentery, syphilis), formation of cicatrices, malignant tumors (carcinoma), and chronic inflammatory conditions of the gut.

(9) Ileus from calculi: (a) By gallstones, more frequent in the female than in the male; usually occurs between the ages of forty and sixty. The diagnosis is based on the previous history and the presence of changes in the liver. (b) By enteroliths. These are most often situated in the ampulla recti or in the recesses made by the sacculum of the colon. The diagnosis of this rare condi-

tion is only possible when the hard concretions are located in the rectum. In some cases the Roentgen ray may disclose their presence. Concretions may result from vegetable sclerenchyma in the residue of undigested fruit or vegetables.

(10) Ileus in consequence of foreign bodies which have gained access into the bowel by way of either the mouth or the anus. This form of ileus must be particularly kept in mind in dealing with the insane. Artificial teeth, needles, pins, nuts, marbles, stones and beads are some of the foreign bodies that gain entrance to the intestine.

(11) Ileus from fecal tumors, a rare occurrence which may supervene in cases of chronic constipation. A fecal mass may attain an enormous size.

(12) Ileus from the accumulation of large numbers of ascarides—quite rare.

III. Paralytic and Spastic Ileus. Paralytic ileus occurs in cases of marked paralysis of the intestine, in severe traumatic injuries, in long-continued grave constipation, after abdominal operations, and in peritonitis and appendicitis. It is explained either by injury to the nerve fibers supplying the muscle fibers of the gut, or by reflex processes. A particularly striking example is afforded by the ileus which occurs when a testicle is retained and becomes inflamed in the abdominal cavity.

Spastic contraction of an intestinal loop, which may become no larger than a finger, supervenes without any known cause reflexly from derangement of the vegetative nervous system (see page 387), in connection with the other forms of ileus and in gastric crises.

Angulations are anatomic throughout the colon; and all will recognize the terms hepatic flexure, splenic flexure, sigmoid flexure, and rectosigmoid flexure. In normal conditions these flexures retard to a slight degree the fecal current, and when through displacement or any other cause the bend is exaggerated the obstruction is proportionately increased (Figs. 106 and 107). Thus in gastroenteroptosis (see Chapter XXX) the transverse colon is carried downward in the abdominal cavity, and unless the ligaments give way the normal hepatic and splenic flexures become more and more acute and obstructive as the colon descends. This accounts for the distention and tenderness over the cecum in such cases. In many instances of this kind the right kidney and the hepatic flexure descend along with the transverse colon, and then only the splenic flexure is accentuated.

If ileus occurs in obese individuals from no assignable cause, acute pancreatitis should be thought of. Frequently there is also pain in the pancreatic region, which may either be spontaneous or elicited on pressure; moderate fever; and transient excretion of sugar.

As an aid to diagnosis in acute intestinal obstruction the stomach tube should be passed from hour to hour and the contents carefully examined. If these are dark brown, with a fecal odor, acute intestinal obstruction may be diagnosed. An early and constant manifestation of intestinal obstruction is acute dilatation of the stomach (see page 486). Adhesions can usually be demonstrated by means of the bismuth mixture and the Roentgen ray. Both the fluoroscopic examination and a number of roentgenograms may be necessary.



FIG. 106. Acute flexure of the sigmoid bound by adhesion to the rectum (Tuttle.)

Symptoms. One of the first symptoms of ileus is abdominal pain, which varies in intensity and is sometimes of intolerable violence, especially in ileus of the small intestine; in contradistinction to the pains of stenosis, those of ileus are more continuous in character. The pain from obstruction of the small intestine is

always located above the umbilicus, while that of the large intestine is always at or below the umbilicus. The pains are usually associated with vomiting, which persists from the beginning until the end of the disease. At first the contents of the stomach are ejected, then the vomitus becomes feculent, and finally absolute fecal vomiting supervenes. This is accompanied by complete

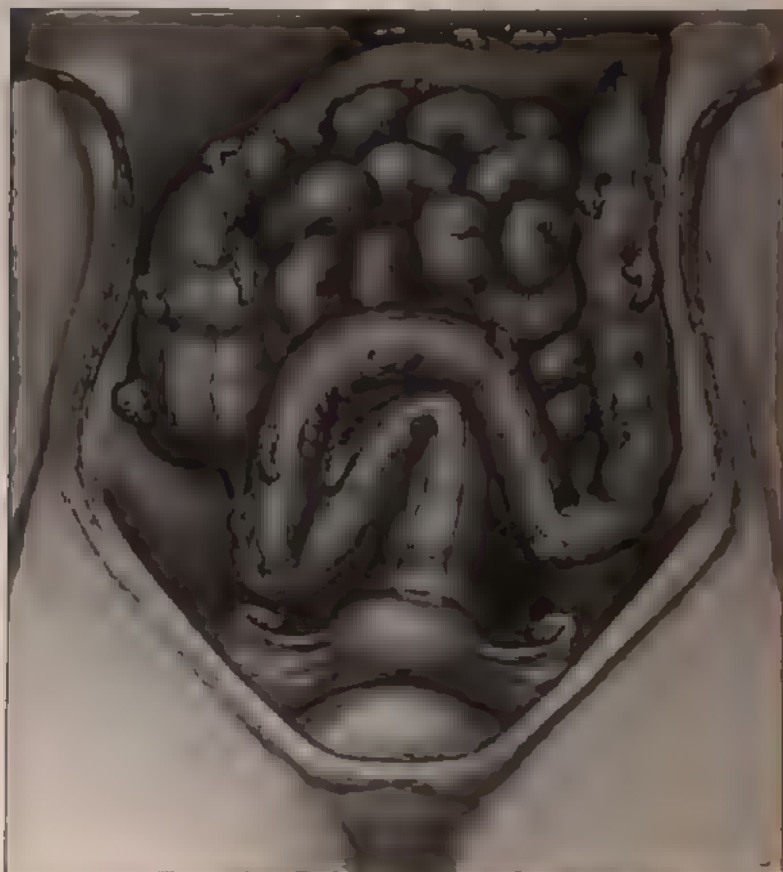


FIG. 107. Acute flexure of the sigmoid bound by adhesion with chronic appendicitis (Tuttle.)

obstipation and the absence of flatus through the rectum. Very soon after the development of the ileus the abdomen becomes greatly distended by meteorism; this is most marked in cases of ileus low down in the large intestine. The whole length of the intestine is usually distended so that the meteorism affects the whole of the abdominal cavity. More rarely there is local meteorism, which when situated in the intestinal loops affected by the

occlusion may become diagnostically important. Differing from stenosis, peristaltic movements are entirely absent in ileus, or only traces may be discovered in a few single coils of the intestine. The general condition of the patient becomes exceedingly grave in a remarkably short time from the commencement of the disease; this is due to the shock caused by the ileus, and is most strongly marked in cases involving the small intestine. The general condition is aggravated by the total loss of appetite, by the impossibility of ingesting food, by extreme thirst, and by intestinal toxemia. To this grave general collapse there is soon added weakness of the heart. The quantity of urine excreted is very small. In the first days of the occlusion of the small intestine large quantities of indican are found in the urine, and this point is of diagnostic value in excluding ileus of the large intestine. The indicanuria is diagnostically useless in the later stages of the disease. Hemorrhages from the bowel take place occasionally; they are especially apt to occur in cases of intussusception, strangulation, gallstone ileus, and volvulus. In volvulus particularly, necrotic perforation and peritonitis are among the imminent dangers. When the symptoms persist without any change, the disease becomes continuously worse and finally causes death, the patient retaining consciousness, in occasional instances, until the last.

The toxemia which develops is due to a primary proteose (Whipple) which may be precipitated by alcohol or ammonium sulphate solution. This poison is easily isolated from closed loops of the intestine and has been found to be very toxic.

Clinicians now believe that death caused by intestinal obstruction is due to the absorption of toxins originating in the epithelium of the duodenum or other parts of the digestive tract.

Treatment.—In a case of ileus the question, after establishing the diagnosis, is, whether a conservative internal treatment is properly indicated, or whether the case should be immediately referred to the surgeon. The possibility of a recovery by means of internal treatment is least favorable or even *nil* in cases of strangulation and internal hernia, intussusceptions, internal strictures, retraction of the mesentery, adhesions of intestinal loops, and ileus caused by compression. All these cases belong *per se* to surgery, but the results from operative measures are best in external hernia. In cases of incarceration and of strangulation a cure by internal treatment is practically impossible; operation should be preferred. Intussusception treated surgically is less often fatal than after internal treatment. The treatment of intestinal obstruction can only be surgical; the results, however, are not satisfactory because of the character of the disease (tuberculosis, carcinoma). Operative intervention offers a poor prognosis in cases of retraction of the mesentery, and in cases of adhesions between intestinal loops subsequent to ulceration (tuberculosis).

The surgical results are often unsatisfactory in all forms of ileus because the patients are transferred to the surgeon far too late. The possibility of benefit bears a close relation to the time of the operation, which should take place within the first forty-eight hours. A patient with ileus rapidly loses his power of resistance to shock, so that operations which are trifling in themselves often result fatally. Intestinal toxemia and septic conditions also play an important part. Furthermore, the results are frequently bad because during the operation complications are discovered which either greatly prolong the time of the operation or render it impossible to remove the obstruction. The vomiting deprives the organism of nutrient materials and water. Normal saline injections supply both water and chlorin to the body and thus prevent desiccation of the tissues. Saline transfusion (hypodermoclysis) should always be practiced in intestinal obstruction pending the decision as to operative measures.

Ileus due to stones or foreign bodies does not offer very good operative possibilities, and may at first be treated by medical means because it is occasionally possible to remove the obstacle in this manner. When this is impossible, it is absolutely necessary to operate.

Volvulus of the sigmoid flexure may reduce itself spontaneously; this generally takes place slowly, and it is proper to wait two or three days for the spontaneous reduction; no internal medication is known that will directly improve the condition.

Pushing massage of the abdomen, when carefully done, is of great help (Fig. 108). The patient assumes the knee-chest position, the physician standing at his left side. The palms of the physician's hands are on the anterior surface of the abdomen underneath. The right hand lies between the umbilicus and symphysis transversely across the abdomen; the left hand likewise lies transversely but between the umbilicus and the ensiform process. The physician exerts alternate pressure above and below, passing both hands in the longitudinal direction of the abdomen. At each push the hands simultaneously approach the umbilicus and move away from it.

Fecal tumors, ascariides ileus, spastic and paralytic ileus must be treated internally.

The field for the activity of the surgeon in the treatment of ileus is thus theoretically very extensive. But unfortunately surgical intervention in cases of ileus is, as stated above, surrounded by so many dangers and complications that in practice, considering everything, medical and surgical treatment may be regarded as about on a par so far as results are concerned. Of course the principle should be held firmly that, when diagnosis is established, operation should follow as soon as possible, before the condition of the patient becomes grave. In those cases also in which internal

treatment is permissible for a time, it is better not to persist in such measures too long. The object of any kind of operation is, first of all, the removal of the obstruction. This may consist in the separating of adhesions, the release of stones within the intestine, the resection of large or small portions of the intestine, or the removal of compressing pathologic conditions. It may happen that it is impossible to perform a radical operation. Then there is the possibility of either entero-anastomosis with exclusion of the diseased portion of the gut, or enterostomy with establishment of an artificial anus. These palliative operations are often very serviceable, and frequently when the general condition is improved the radical operation may be attempted later.

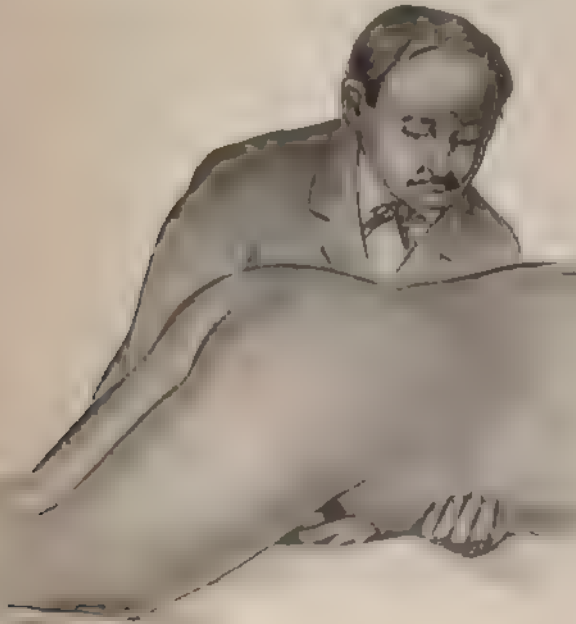


FIG. 108. Pushing massage of the abdomen for volvulus. (Zakusowski-Egger.)

In the internal treatment of ileus the primary indication is to counteract the rapid failure of the patient's strength, and to this end dietetic measures are, of course, of greatest importance if at all practicable. It must be our constant endeavor to introduce into the body the greatest utilizable number of calories; and for guidance we should know whether the ileus affects the large or the small intestine. In cases of ileus of the small intestine, nutrition by mouth is often impossible because of the absolute loss of appetite and the continuous vomiting. In such cases it is not at all desirable

to administer much by the mouth, since, even if it were retained, it would add a new burden to the intestine. Rectal alimentation should preferably be freely employed. In cases in which nutritious enemata are retained badly, subcutaneous nutrition may be employed in the form of transfusion of solutions of protein, sugar or fat, or percutaneous nutrition by means of inunctions of fat into the skin. Generally speaking, very little success will be attained by this method of feeding, but it is possible that in some few cases subcutaneous nutrition may contribute slightly to the sustenance of the bodily strength and may therefore be justified as a last resort. Then, too, the patient's spirits are kept up by the idea that something is being done for him. In cases of this kind, with low nutritional conditions, physiologic salt solution (0.9 per cent.) should be administered either subcutaneously or by proctoclysis. By this means the paucity of water in the organism is counteracted, and the water introduced assists in eliminating toxic substances from the body by directly stimulating diuresis. When the blood-pressure is rapidly decreasing during the first acute stage, the intestine not having yet become paralyzed, some surgeons recommend intravenous transfusion of normal saline solution with the addition of a few drops of epinephrin solution.

Conditions are quite different in ileus of the large intestine. Here it is possible for the upper portions of the gut to partly digest and absorb some of the proper food. Food should therefore be given in as large quantities as possible, but the diet should consist of liquids only, or liquids with the occasional addition of semi-solids. The following are suggested: milk, cream, beef tea, beef jelly, meat juice, beef tea preparations containing protein, liquid egg dishes, gruels and soups with flour, legumes, and addition of butter and eggs. All these foods should be given frequently and in small portions. When there is a tendency to vomiting, the milk should be given cold, but this is inadmissible when there is severe colicky pain. The administration of small quantities of alcohol, pure or as an addition to suitable foods, has a favorable action on the heart. Abundant quantities of liquids by the mouth, subcutaneously, and per rectum, are indicated. Also feeding by rectum should be instituted if practicable (see page 243).

The care of the strength of the patient being provided for by these methods, the question arises: What are the important remedies at our disposal for directly attacking the ileus? Lavage of the stomach may act favorably, particularly in ileus of the small intestine, and possibly be the means of saving life. The lavage should be done quite early in the attack, especially when feculent vomiting is a feature of the case; if the decomposing materials above the obstruction can be removed, as is sometimes possible, by means of gastric lavage, the stagnation is relieved and the excessive peristaltic movement diminished. Another effect is the

reduction of pressure above the closure—which may mean the removal of an obstacle to the spontaneous overcoming of the obstruction. Lavage of the stomach should, as a rule, be instituted during the first forty-eight hours, but when the general condition is satisfactory it may be postponed somewhat longer. When the heart is alarmingly weak and the patient very low, lavage may sometimes turn the scale in favor of recovery; at this stage, however, it severely taxes the remaining powers of resistance, and under some circumstances hastens the fatal issue. The washing-out of the stomach must be done several times daily (two to five times), or at least twice a day. Each single lavage must be continued until the water returns clear, and the stomach should be left quite empty. The next washing is always made at a time when the stomach again becomes distended, indicating a regurgitation of new material into it.

If necessary, morphin, 0.01 Gm. ($\frac{1}{4}$ grain), should be given subcutaneously prior to the lavage. In the presence of great weakness, camphor or caffein, 0.1 Gm. ($1\frac{1}{2}$ grains) subcutaneously, is indicated.

As stated above, the question of the utility of lavage arises particularly in cases of ileus of the small intestine. Its great efficiency is well known in arteriomesenteric contraction in consequence of acute gastric dilatation (see page 486). It usually fails in cases of volvulus, intussusception of the large intestine, internal strictures, stones, fecal tumors, and paralytic and spastic ileus.

Sometimes rectal injections—which can only be considered in ileus of the large intestine—do very well. The quantity of water introduced may act in an altogether mechanical way on the obstruction by virtue of the pressure it exerts on intussusceptions; it may loosen foreign bodies and stones; it may reduce a volvulus, soften fecal tumors, and in paralysis of the intestine it may stimulate peristalsis. Water enemata with soap and oil are the best for this purpose.

When it is intended to employ strong pressure upon the point of closure, it is best to allow two or three liters (quarts) of normal saline solution to flow in by means of a long rectal tube, strongly compressing the anus manually, the patient meanwhile maintaining the knee-chest position. When larger volumes of water are used, care should be taken to see that all of the water returns before a renewal of the injection is attempted. Otherwise it is easily possible for the large intestine to become overdistended, with resulting perforation.

Nothnagel has recommended the employment of ice-water, and the use of the siphon (Fig. 34) by which ice-cold water containing carbon dioxid is allowed to pass in through a rectal tube under high pressure.

Good effects have been reported from the employment of ether:

70 Cc. (3ij $\frac{1}{4}$) of ether with 300 Cc. (3x) of water is made to flow into the rectum through a soft-rubber rectal tube. These injections are to be repeated several times daily, until flatus and feces are discharged.

The patient should always be given the benefit of extensive irrigation of the bowel before surgical intervention is resorted to. Not infrequently, in cases that look hopeless, evacuation is accomplished in this way. For this purpose I have found the supernatant oil apparatus (Fig. 109) invaluable. This is a glass bulb (*b*) with two lateral openings, to one of which a short soft-rubber rectal tube (*a*) is attached, and to the other an ordinary rubber-bulb syringe (*c*). The apparatus is easily improvised; any glass bulb with two openings will answer. The bulb is filled with olive oil (it should hold about 250 Cc., or half a pint), and the free end of the syringe is inserted into a basin (*d*) containing two liters (quarts) of water. On pressing the bulb (*c*), the water forces the oil into the intestine. On account of the lighter specific gravity of the oil, it precedes the water through the coils of the colon. This method is more likely to produce an evacuation than the use of either water or oil alone. I rarely call for the aid of the surgeon before I have used this apparatus.



FIG. 109.—Supernatant oil apparatus. *a*, rectal tube; *b*, glass container for oil; *c*, rubber bulb syringe; *d*, basin for water.

The distention of the intestine with air serves the same purpose. The liberation of intussusceptions has often been observed after this procedure; and foreign bodies and stones may likewise be loosened, and angulations straightened out. Insufflations with air are contra-indicated when an ulcer is suspected, and in cases of necrosis, hemorrhage or peritonitis.

The Noble¹ enema is highly recommended when there is difficulty in obtaining a movement of the bowels:

	Gm. or Cc.	
R—Magnesii sulphatis	60 0	3ij
Glycerini	60 0	3ij
Olei terebinthinæ	15 0	3ss
Aquæ q. s. ad	240 0	3viij
Misce.		

Sig.—Use as an enema and retain as long as possible.

When fecal tumors are located near the anus, the rectum may have to be cleared out manually, and bile enemata are particularly efficacious (see page 226).

¹ This formula was first published by C. P. Noble, of Philadelphia and recommended by him for producing the most positive results in paralytic bowels.

Occasionally recovery may result from puncture of the intestine as recommended by Curschmann. This procedure is not recommended unless surgery is absolutely contra-indicated. In such cases, after puncture, there is a diminution in the tension in the intestinal loops, in consequence of which the incarcerated portions of the gut can more readily return to the normal position. The puncture is performed in the following manner:

A long cannula (provided with a stopcock) of the size of a Pravaz syringe needle is carefully sterilized and, the stopcock being closed, is inserted into an intestinal coil. The cannula is then connected with a rubber tube, and this is conducted into a bottle filled with a watery solution of salicylic acid, which is inverted in a basin containing a similar solution. When the stopcock of the cannula is opened the intestinal gases pass into the bottle, at first in one continuous stream and later on more slowly. Meanwhile the cannula must not be held tightly; its guidance is to be left to the intestine. Punctures may be made at various points and should be frequently repeated. Puncture of the intestine may be undertaken in cases of strangulation and of volvulus, but surgery is better. It is strictly contra-indicated in intestinal paresis, in intussusception, in peritonitis, and when gangrene is suspected.

A few physicians have recommended massage of the abdomen, but as a rule this can only be considered in cases of ileus due to fecal tumors. At any rate it is never to be employed without the exercise of the utmost caution, because of the danger of peritonitis and of perforation.

Warm applications and Priessnitz bandages to the abdomen are symptomatically pleasant (see page 250).

Medication.—Purgatives (see Chapter XIV) are permissible in ileus due to fecal tumors, for softening the hard fecal masses and for assisting their passage by stimulation of the peristaltic movements. In ileus paralyticus castor oil may be given, and its action assisted by enemata. In all other cases of ileus, purgatives act injuriously by increasing the peristaltic movements which are already excessive, thus rendering the removal of the obstruction more difficult. On the other hand, some anodynes have a beneficial action (see page 274). First of all, opium. This drug relieves the pains, acts as a general sedative, diminishes shock, and induces sleep. It also lessens the vomiting and quiets the convulsive contractions of the intestine. Consequently opium favors the possibility of a spontaneous liberation of the obstruction. It also acts as a prophylactic against local peritonitis, which is likely to occur when the ileus has existed for some time; by its antispasmodic action the opium prevents traction upon the inflamed peritoneum. Opium is, however, indicated only in the first stage of ileus, and not then when the heart is weak or the general depression extreme. Nor should one forget that the sense of well-being experienced by the

patient as a result of the administration of opium may conceal the true state of affairs, making the case appear to be much more favorable than it actually is. It is well, therefore, to limit the use of this drug, that an unbiased view of the case may be obtained.

In the past ten years rather pleasing results have followed the administration of atropin in ileus. It is well known that atropin in small doses relieves vagotonia and has a stimulating effect upon the muscular elements of the intestine, and therefore serves to liberate spastic ileus and spastic conditions of the intestine due to mechanical obstruction. It is necessary from the very beginning, however, in such cases, to employ large doses—doses which exceed the official maximum. If the patient has taken opium previously, 0.005 Gm. ($\frac{1}{2}$ grain) of atropin sulphate should be given without any delay, and this dose should be repeated if no improvement shows itself after twelve hours. The opium antagonizes part of the atropin, so these large doses do not induce any serious symptoms of poisoning. If no opium has been given previously, the initial hypodermic injection of atropin should be 0.002 Gm. ($\frac{1}{80}$ grain), and after twelve hours another dose, of 0.005 Gm. ($\frac{1}{2}$ grain). If, twenty-four hours subsequent to the giving of atropin, no flatus has been expelled, it will hardly be advisable to push this medication further. Possible symptoms of toxemia, delirium, hallucinations or convulsions are counteracted by morphin (see page 275).

Occasionally physostigmin also may be given, with great caution, thus: 0.001 to 0.002 Gm. ($\frac{1}{80}$ to $\frac{1}{40}$ grain) physostigmin salicylate, or 0.001 or 0.01 Gm. ($\frac{1}{80}$ to $\frac{1}{8}$ grain) physostigmin sulphate (see page 283).

Effects similar to those of atropin and physostigmin may apparently be produced by pituitary extract or by the peristaltic hormone (see page 667) which may now be considered a valuable addition to the internal remedies at our disposal for the treatment of this form of ileus. It induces a peristaltic motion approximating the physiologic more closely than that induced by physostigmin.

CHAPTER XLVII.

STRICTURES OF THE INTESTINE.

STRICTURE of the intestine consists of abnormal narrowing of the bowel, from either cicatricial contraction or the deposit of adventitious tissue.

Strictures or stenoses of the duodenum or the jejunum are called "high," while those that occur at any point from the beginning of the ileum onward are described as "low" or "deep" strictures.

STRICTURES OF THE SMALL INTESTINE.

Symptoms.—Strictures situated just below the pylorus and above the papilla of Vater (suprapapillary stenoses) generally induce the same symptoms as stenosis of the pylorus. The most prominent symptoms are referable to the stomach, particularly vomiting of the stomach contents. In such cases the correct diagnosis is usually difficult to arrive at, and stenosis of the pylorus is assumed to be present (page 490). A frequent cause of this form of stricture is wedged-in gallstones, which are too large to find their way into the gut but are forced by the bile and the natural expulsive effort of the bile duct into a "corner," so to speak. Furthermore, adhesions and strictures subsequent to duodenal ulcers and carcinoma may possibly be present.

Stricture situated below the papilla of Vater (infrapapillary stenoses) but in the duodenum also induce gastric symptoms, such as distention, pressure, pain, eructation, nausea and vomiting. But in these cases the vomited matter does not consist of pure gastric contents, for the regurgitating chyme is mixed with the digestive secretions of the intestine, the pancreatic juice and the bile, and is discolored by the latter. The finding of such intestinal secretions in the stomach contents may be of high diagnostic value.

The vomiting, as in stenosis of the pylorus, may continue after the ingestion of any food, because there is a continuous pressure backward in consequence of the stricture being situated so short a distance below the stomach. Accentuated peristaltic movements are not usually found in these strictures, because the liquid chyme is able to pass through the stricture or escape back toward the stomach. This also accounts for the absence of colicky pains. The bowels are usually moderately constipated, and the feces may be light in color in consequence of the absence of biliary coloring

matter. The urine may be rich in indican. The appetite quickly decreases as the stagnation persists; the body weight fails, and the percentage of water is greatly diminished. The stomach itself may be either normal in size or dilated; during the early stages of the disease it may perform its secretory functions quite normally.

When the stenosis has existed for some time, *sarcinae* and long bacilli are usually present. When palpation reveals a tumor in the duodenal region, this is very likely a carcinoma. When palpation is negative, the previous history is of importance; if gallstone colics have occurred, attention should be given to the possibility of an occlusion by gallstones, or of adhesions—often found in females. In men it may possibly be a duodenal ulcer which is causing the stricture. The diagnosis of probable carcinoma is apt to be correct in advanced life. Other affections to be kept in mind are: lymphomata, sarcomata, compression by metastatic tumors, diseases of the pancreas (cysts, tumors, adipose tissue, necrosis), and retroperitoneal tumors.

Stricture of the jejunum induces gastric symptoms. Vomiting is particularly frequent, but does not take place as often as it does in stricture of the duodenum, for the reason that there is more space above the stenosis for the collection of the chyme. The vomiting, when it does occur, is more copious than in duodenal stricture. When the jejunal stricture is situated far down, the vomited matter gradually assumes fecal characteristics and contains bile.

Stricture of the Ileum. The intestinal symptoms predominate in this condition. The vomitus becomes fecal. Peristaltic movements are both visible and palpable, and colicky pains may develop, particularly in cases of carcinoma. The intestinal symptoms become more pronounced in proportion to the relative proximity of the stenosis to the cecum. As a rule, however, marked symptoms appear rather late—only during the more advanced stages of the stricture—and are preceded by vague disturbances of bowel function, such as constipation or diarrhea or alternation of both. Occasionally multiple strictures occur simultaneously.

The causes of jejunal and iliac strictures are adhesions, kinks and angulations caused by gastroenteroptosis (see Chapter XXX) or inflammation of the female genital apparatus or of the appendix, and hernias. Cicatrized tuberculous ulcers are rarely the origin of strictures. Syphilitic strictures are very rare. Carcinomata occur in elderly people and in people of still more advanced years. Stricture of the small intestine can often be made out by means of the Roentgen ray. The bismuth shadow shows a persistent stagnation for many hours. Loops of the intestine may appear dilated either as abnormally long, band-like shadows, as broad as the colon, or as ampulla-like hollow spaces as large as an orange, or even as large as a melon, filled with fluid or gas. Either of these conditions is suggestive of obstruction in the intestine (see page 148).

STRICTURES OF THE LARGE INTESTINE.

Symptoms.—In this class of cases the symptoms generally develop slowly. The first sign is constipation, which may persist for months or even years. Constipation should arouse suspicion when it is found in people of mature age and when it resists treatment with mild purgatives so that finally the strongest drastic medicaments are resorted to. To constipation are gradually added colicky pains, to which slight attention is at first given, but which are of great weight in the diagnosis. Should vomiting begin, the suspicion of stricture grows. Chronic distention with gases is a very ominous objective sign. The meteorism is least marked in strictures of the rectum, and more so in those located at higher levels of the large intestine. Increasing meteorism is a sign of the presence or commencement of paralysis of the gut. Of more importance than meteorism is the discovery of visible peristaltic movements in connection with constipation and colicky pains.

The typical course of these spastic peristaltic motions is from an imperceptible start, accompanied by marked pains, to a rigid and forcible contraction of the particular section of the gut affected, and finally a sudden relaxation with loud gurgling sounds in the abdomen. This peristalsis may affect either circumscribed or extensive sections of the intestine. When the abdominal walls are thin the motions are readily perceived with the unaided eye.

One of the most important symptoms of stricture of the bowel, and at the same time the most important sign of carcinoma, is the occurrence of palpable and visible contractions. It is difficult in many cases to tell whether these movements are in the small or in the large bowel. Occasionally a distinction can be made by examination of the vagina during the attack of pain when the movements are very active. Oftentimes the site of the obstruction may be suspected by the discovery of a splashing sound in the bowel just above it. One peculiar sign, which is practically pathognomonic of stenosis of the bowel, is the sudden appearance of small coils of bowel which vanish very quickly and reappear. The section of intestine leading to the obstruction will often be found stiffly contracted.

A stricture of the large intestine can usually be made out by the use of the Roentgen ray (see page 148). The bismuth mixture should be introduced by rectal injection while the patient lies on his back. The upward passage of the mixture can be kept under direct inspection with the fluoroscope. In the study of constrictions of the large intestine, fluoroscopic examination during the flow of the bismuth mixture through the colon is more valuable than roentgenograms. The former enables us to study the motility of the intestine, while the latter show only single momentary phases of the position of the filling colon. In the presence of constrictions

the fluoroscopic examination shows that the bismuth stops flowing at the site of the obstruction. There is usually a bulging at this point, and suddenly a finger-like process of bismuth passes onward. It is possible in this way to differentiate between tumors, spasms and adhesions as the cause of the stricture. Anomalies of position, formation of loops, dilatation and stenosis of the lumen, and diverticulosis, can all be easily recognized.

Strictures of the large intestine may be caused by adhesions (gynecologic diseases, former operations) and subsequent torsion, or by cicatricial formations (tuberculosis, dysentery, syphilis) involved in the previous history; but the most frequent cause is carcinoma.

When the stenoses persist for some length of time, they may give rise to the formation of ulcers of distention and stercoral abscesses. The stenotic portion of the intestine may also ulcerate on the interior wall, especially in the presence of malignant tumors. When this is the case blood and pus are often found in the feces in large quantities, so that an examination of the stools at first seems to point to dysentery.

Treatment of Intestinal Strictures.—Strictures of the intestine that are due to gallstones or swallowed foreign bodies are often manageable by internal treatment. Other intestinal strictures can be cured only by operative treatment; and this must be the aim of any sort of therapeutic intervention. The question then arises: Which are the cases suitable for operation? Without any reservation, all cases of malignant stenosis (carcinoma, tuberculosis), even if as yet no evil effects seem visible, belong to surgery; also all those cases of benign stenosis, accompanied by grave symptoms, in which every kind of internal treatment has been in vain. Notwithstanding the splendid technic of modern abdominal surgery, poor results are often obtained because the patients are very anemic and cachectic—unable to survive the operation. In the early stages of the disease most patients, unfortunately, cannot be convinced of the necessity of an operation. Later it may be impossible to operate because of the great weakness of the patient, or after opening the abdomen such complicated anatomic relations may be disclosed as to make a radical operation impossible or even forbid a palliative operation. Matters assume the worst aspect in this respect in cases of stenosis of the pars superior duodeni, in which a radical removal is usually impossible. There may be some hesitation as to whether it is advisable to transfer to the surgeon cases of benign stricture which do not cause any grave symptoms. As a matter of principle it will probably be correct to advise operation, as it cannot be predicted in any case whether the stenotic trouble will remain stationary or become worse. An exacerbation of the whole condition may sometimes arise quite suddenly. But one may be justified in undertaking internal treatment at first, so long as it is certain that the stricture is not of a malignant character.

The internal treatment is directed to:

1. Curable strictures (calculi, foreign bodies).
2. Benign strictures with slight symptoms.
3. Malignant strictures in which operation is refused by the surgeon.

In regard to the diet, the following general rules apply to all cases of intestinal stricture. The diet must be of such a nature that it will pass the stricture—that is to say, absolutely liquid in grave stenoses, and semisolid in cases of slight stenosis. It should also be antiputrefactive. The feedings should be only large enough to provide the necessary number of calories. These directions must be insisted on most rigidly in stenoses of the small intestine. In strictures of the large intestine the diet may be somewhat more liberal, with more variety, and coarser (see page 174).

In stenoses of the small intestine the chief article of diet must be milk, which should be made antiseptic by the addition of salicylic acid (see page 176). Kefir, yoghurt, gruels and mucilaginous soups are permissible. Various leguminous flour soups, with additions of scraped meat, mashed vegetables, artificial albuminous preparations, and plenty of butter, oil and cream, apple sauce, mashed potatoes, well-softened wheat bread, zwieback and biscuits, may be given.

The diet in strictures of the large intestine differs. Meat may be given well cut up, and an attempt should be made to obtain, if possible, a slightly purgative effect by utilizing naturally purgative articles of food, such as fruit juices, honey, fruit acids, and jams of various fruits. Great stress must be laid on the fruit being of good taste, flavor and variety, delicacies being used freely. The articles altogether prohibited are: fresh fruit, boiled skin and seed fruit (the seeds of fruit are particularly dangerous), tendinous meat containing much connective tissue, coarse vegetables, and potatoes. The careless use of such coarse articles of food occasionally causes complete closure of the stenosis and other grave complications.

When the nutrition by mouth proves inadequate, careful rectal feeding must be instituted (see page 243).

Strictures of the small intestine situated high up demand, like stenoses of the pylorus, regular lavage of the stomach, according to the principles laid down for stenosis of the pylorus. Rectal injections are required in all cases in which constipation is present or in which it alternates with diarrhea, in order to avoid the administration of purgatives. In such cases rather voluminous injections (one liter) are to be used, or small-sized enemata retained for a long time. When the strictures of the colon are situated low, medicated lavage or irrigation may be very useful, especially when the stenosis is ulcerating, secreting much pus, and gives rise to hemorrhages. In such cases the diseased portion is to be cleansed very gently with infusion of chamomile and subsequently with a

solution of borax or a suspension of bismuth subnitrate. When bismuth subnitrate is introduced by irrigation it is frequently possible to keep the formation of pus and mucus and the loss of blood within moderate limits, thus favoring the general well-being of the patient (see page 752).

Medication.—The purgatives are the medicaments particularly to be considered here, and of these the milder ones are to be employed, such as castor oil, cascara sagrada, phenolphthalein, tamarinds, and the bitter mineral waters, in rotation but continuously (see page 284). In cases of stricture of the large intestine with rigidity of the intestinal walls the anodynes are frequently very effective and prompt purgatives, especially opium and extract of belladonna in the form of suppositories (see page 274). The severe pains often require the above-named anodynes, and also morphin. In hopeless malignant cases, warm applications to the whole of the abdomen are grateful. When the suspicion is well founded that peritoneal adhesions are the cause of the stenosis, fibrolysin treatment may be given a trial (see page 484).

Strictures due to calculi and foreign bodies should be treated by means of purgatives. Such foreign bodies and calculi, when located in the large intestine, can frequently be loosened by skilfully applied irrigations, and may be dissolved by appropriate irrigating fluids (see Chapter XI).

Liquid petrolatum has been used to lubricate the mucous membrane of the intestine and thus assist the gliding along of the feces. It passes through the whole alimentary canal unchanged, and frequently affords relief in cases of slight stenosis. I have seen the marked peristalsis abate and normal movements ensue when tablespoonful doses were given three times a day (see pages 650 and 664).

When it is impossible to relieve these strictures by internal medication, it becomes absolutely necessary to resort to surgical

■ ■ ■ ■ ■

CHAPTER XLVIII.

TUMORS OF THE INTESTINE.

CARCINOMA; SARCOMA; LYMPHOSARCOMA; ADENOMA; POLYPI;
LIPOMA; MYOMA.

MALIGNANT NEOPLASMS OF THE INTESTINE.

Carcinoma.—The frequency of the occurrence of carcinoma of the intestine is shown by statistics. According to a statistical statement of Heimann, compiled from the hospitals in Prussia, there were, among a total of 20,544 cases of carcinoma, 1706 cases of carcinoma of the intestine and 4288 of carcinoma of the stomach.

Maydl reports that of 41,838 necropsies made in the Pathologic Institute at Vienna during twenty-four years, alimentary carcinomata were present in 3585 cases, nearly 10 per cent. The intestine was involved in 343 cases as follows: the rectum in 162, the colon in 164, the ileum in 10, and the duodenum in 7. It is a fact, however, that the duodenum becomes carcinomatous almost as frequently as the jejunum and the ileum together. In 21,624 necropsies, Zemann found 165 carcinomata of the intestine, 8 per cent. of the total number.

Carcinoma is most frequent in the large intestine, and of these cases, according to Leube, 80 per cent. are of the rectum (Plate XXVIII); then follow, in order of frequency, the sigmoid flexure, the colon (particularly at the flexures), and the cecum. Carcinoma of the small intestine is comparatively rare, but the duodenum is more frequently involved than any other part of this region. Female and male are attacked in about equal proportions, but so far as carcinoma of the rectum is concerned the male sex predominates. Intestinal carcinoma occurs most frequently between the ages of forty and sixty. Occasionally it has been observed in early life and even during childhood.

While carcinoma is said to be uncommon in some countries, in Japan there are 25,000 deaths a year from the disease; in England it is estimated that, of persons over thirty-five years of age, one out of every eight women and one out of every eleven men die of carcinoma, a greater death-rate for the age period than from tuberculosis; and we are not far behind in this country, with 80,000 cases constantly in progress and over 40,000 deaths each year from the disease.

Holländer gives some interesting family histories from cases of carcinoma of the intestine, which show a most striking family tendency to carcinoma in some form, chiefly of the gastro-intestinal tract. In the first case the husband and wife (who had indistinct histories of carcinoma), a son and a daughter died of carcinoma; in the next generation four children died of the disease, in the next generation two, and in the next three. In another instance reported, one case occurred in one generation, four in the next, and two in the next ("generation" including the collateral branches of the family as well as the direct descendants). He considers the family history a very important point in the diagnosis of carcinoma of the intestine, since it seems to be intestinal carcinoma in which these striking family histories are most commonly met. Another point which he considers of much importance is the occurrence of skin changes, chiefly in three varieties: (1) vascular changes; (2) seborrheic warts; and (3) pigmental patches. The vascular changes consist in the appearance of large numbers of small spots, from the size of a pinhead to that of a pea, which have the appearance of small angiomas. The warts are likely to occur in very large numbers and may reach an unusual size. He has seen them as large as pigeon's eggs. The pigmented areas vary in appearance and depth of color, but may involve practically the whole body. These signs he has found very common in intestinal carcinoma.

Pathology.—The most frequent form of carcinoma is that which consists of cylindric epithelial cells originating from the cylindric epithelium of the mucous membrane and showing a glandular structure. Then follows the medullary carcinoma, which has a tendency to disintegration and ulceration. The gelatiniform carcinoma (colloid) is rare, and its most usual location is in the rectum. Scirrhus is very rare. Intestinal carcinomata, as a rule, give rise to few metastases.

Carcinoma of the Small Intestine.—Carcinomata of the small intestine are rare as compared with similar growths of the large intestine. The carcinomata of the duodenum are classified as suprapapillary, infrapapillary, and papillary. The location of the duodenal papilla (Vater), an elevation near the point where the ductus communis choledochus enters the duodenum, is used for this classification.

If a tumor is present in the suprapapillary section of the duodenum, it lies to the right of the median line, between the lower border of the ribs, the navel and the gall bladder. A tumor thus situated will be found, on palpation, to be easily movable. If the growth arises in the infrapapillary section of the duodenum, it will lie in the same position, but it will be slightly if at all movable, being firmly fixed by the pancreas and the peritoneum. The common bile duct and the pancreatic duct perforate the median

border of the papillary section of the duodenum. If carcinoma develops here, the opening of the bile duct will be closed and jaundice will result (see page 614). Growths in the first portion of the duodenum are similar to those associated with ulcer of the stomach. Those of the papilla are, as a rule, cylindric-cell adenocarcinomata. Those of the prejejunal region tend to be broad, flat and ulcerated, and to form stenoses. The duodenal contents removed with the duodenal tube show a deviation from normal of the pancreatic ferments (see page 103).

Symptoms.—The symptoms of suprapapillary carcinoma are: pains in the region of the stomach, pressure, nausea, increasing emaciation, lassitude, and the appearance of a tumor in the right hypochondriac region. When the tumor, as a consequence of increasing growth, narrows the passage, the result is duodenal stenosis. It is sometimes scarcely possible to establish a differential diagnosis between duodenal stenosis of this character and one of different origin. Suprapapillary and intrapapillary carcinomata offer very similar appearances. But in the case of the latter the vomited matter is stained by bile. It is only possible to diagnose such a case when a tumor is palpable, and even then the difficulties in the way are very great. Symptoms of stricture are also manifested in this disease, especially toward the end.

Of particular importance respecting papillary carcinoma is the development of the chronic jaundice without demonstrable disease of either the liver or the pancreas.

Carcinoma of the duodenum is usually of the same type as carcinoma of the pylorus. The growth is derived from a simple form of secreting cell, cylindric epithelium, and it forms a new growth of cylindric cells arranged in a manner somewhat similar to that of the normal tissue from which it springs. At the pylorus the glands are formed of a single layer of cylindric cells on a basement membrane; they are situated in the mucous membrane. At the junction of the pyloric end of the stomach and the duodenum the glands pass down through the muscularis mucosa into the submucous tissue, and in the duodenum they become Brunner's glands. The pyloric glands and Brunner's glands are identical in structure, and are continuous with one another, the only difference is as to situation, one being in the mucous membrane, the other in the submucosa.

Diagnosis.—For therapeutic reasons the diagnosis of carcinoma of the duodenum should be made as early as possible—a task that requires the keenest medical acumen. In the majority of cases the tumors are quite large when discovered, for we do not get many symptoms so long as the lumen of the intestine is patulous.

An anatomic peculiarity of intestinal carcinoma is its tendency to develop in ring form and to traverse the inner circumference of the intestine. This peculiarity is frequently manifested, and in

many cases very marked. Such circular formation reduces the lumen of the intestine, and the rapid growth and tendency to contraction lead to a stricture that calls for surgical intervention. In fact, carcinoma of the intestine manifests itself by the phenomena of stricture (see Chapter XLVII).

In the male approximately two-thirds, and in the female one-fourth, of all carcinomata develop in the alimentary canal, while one-half of all carcinomata of the female are in the reproductive organs. It seems an established fact that carcinoma of the duodenum, while very rare, usually originates from a previous old duodenal ulcer.

Carcinomata of the jejunum and ileum are also rare and always difficult to diagnose, particularly if, as is frequently the case, no tumor is palpable. Of diagnostic importance in regard to the presence of a tumor are its mobility, the symptoms of stenosis of the small intestine, and hemorrhage from the bowel. The Roentgen ray is frequently of great help, as are also serologic reactions (see Plate XVII, Fig. 3, and page 543).

Carcinoma of the Colon.—*Symptoms.*—Particular mention must be made of the fact that among the subjective symptoms of carcinoma of the colon intestinal pains are most conspicuous. There may be a permanent dull pain localized at a fixed spot, or paroxysmal abdominal pains, colicky in character, which are very characteristic and may persist for a long time, even years, before the development of any other sign of the disease.

These painful attacks are synchronous with constipation and emesis. The more intense the pains, the more obstinate the constipation. The painful attack ceases after a free evacuation of the bowel. Occasionally there is diarrhea, especially after a constipated period of several days' duration. Vomiting occurs early, only the stomach contents and mucus being ejected; stercoraceous vomiting occurs less frequently. In carcinoma situated very low down, particularly rectal carcinoma, frequent tenesmus is the most prominent symptom.

Diagnosis. The objective signs important for diagnostic purposes are: the presence of a tumor (which is found in 40 per cent. of all cases), meteorism, visible peristaltic movements, rigidity of the intestine, macroscopic blood from the bowels, occult hemorrhage in the feces, diarrhetic stools with blood and pus, anemia, emaciation and cachexia. In some few cases all these criteria may be absent; the carcinoma may be entirely latent and either terminate fatally without being discovered or induce a sudden attack of ileus. A marked loss of weight (forty or fifty pounds) in a patient over forty years of age, without localization of symptoms, is always suggestive of hidden carcinoma of the intestine. The Roentgen-ray examination is of great assistance in the diagnosis. Carcinoma of the large intestine may be complicated with perforation, adhe-

sion and communication with the abdominal organs (stomach, bladder), or perforation through the abdominal parietes or into the retroperitoneal tissues with formation of abscess (Plate XX, Fig. 1).

Attention should be called to a group of cases which are frequently mistaken for malignant growths. Under normal conditions we are able sometimes to feel the cord-like descending colon or the sigmoid flexure, which may be tender to deep palpation, when the abdominal walls are thin and relaxed. The cord-like feel normally disappears when the colon is emptied or inflated. When there are no symptoms of any intestinal disease it is not difficult to diagnose the character of such a tumor-like resistance. It is entirely different when there is a spastic contraction of the colon simultaneous with other symptoms, such as constipation, irregular movements, pain, and disturbance of the general condition. Under such circumstances we are likely to suspect a malignant growth when the condition is one of spastic constipation (see Chapter XXXVII). Such spasms occur as a pure neurosis.

Treatment.—Carcinoma of the intestine cannot be cured by internal remedies; a radical recovery can only be brought about by surgical treatment. Operative therapeutics, however, is often unsuccessful, for a variety of reasons. The diagnosis is usually made too late, at a time when the carcinoma has attained a large size, metastases have developed, and operative removal is no longer possible. Operation is often prevented by the objections offered by the patients and their friends, although the diagnosis may have been established in time. Moreover, the radical operation is frequently a severe proceeding, and the patient, already considerably weakened, is unable to survive it.

The *radical operation* therefore is *a priori* contra-indicated in the presence of marked cachexia and anemia, and when, as shown by the clinical examination, metastases, adhesions of the tumor and other complications are present. In these cases a *palliative operation* is indicated, especially when symptoms of stenosis and ileus have appeared. The radical operation consists in total resection of the carcinomatous portion of the gut. The palliative operation is the establishment of an artificial anus; the radical operation, separation of the diseased portion of the gut by resection of the intestine above and below the carcinoma, and joining of the intestinal ends by anastomosis. The mortality in the resections is 50 per cent., with about three years of life after the operation. Some permanent recoveries have followed the radical resection.

At the International Congress of Medicine held in London, 1913, Bastianelli of Rome read a paper on the operative treatment of malignant disease of the large intestine, in which he gave the following summary, the cases having been collected from the data of a few surgeons of wide experience. Total number of cases operated on more than three years ago, 239; operative cures, 140, alive and

well three years after, 68—42.8 per cent. of the survivors from the operation, or 28.8 per cent. of all cases operated upon.

The *internal treatment* coincides in all its essential points with the therapeutics adopted in cases of stricture and of ulcer. The aim is to increase the disease-resisting powers of the patient as much as possible by dietetic measures and to render life tolerable by mitigation of the pains. The former object is attained by a diet arranged in accordance with the situation of the carcinoma (large or small intestine); the latter by the administration of anodynes and narcotics. Lavage of the stomach and irrigation of the intestine are sometimes indicated.

Sarcoma and Lymphosarcoma of the Intestine.—In contradistinction to carcinomata, *sarcomata* and *lymphosarcomata* of the intestine are of very rare occurrence; they are found particularly between the ages of ten and forty. As a rule they are situated in or on the small intestine or the rectum.

In respect to the pathologic anatomy, all kinds of sarcomata may be present. The ones most frequently found are the round-cell and spindle-cell variety. They originate from the submucous or subserous coat and are generally smooth tumors. They are distinguished from carcinoma by their very rapid growth, and may develop into tumors of enormous size.

The starting point of lymphosarcoma is in the lymphatics of the intestine. Like sarcomata, these growths often cover extensive areas of the intestinal tract, and in consequence of the tissue metamorphosis the affected portion of the intestine becomes a rigid tube, of large lumen, without stenosis.

Symptoms.—Clinically, sarcoma or lymphosarcoma may be suspected when no symptoms of stenosis are found though large tumors are present. The cachexia supervenes more rapidly than in carcinoma; edema, ascites, and metastases in the peritoneum are also sooner developed. The lifeperiod of the patient after sarcoma or lymphosarcoma makes its appearance is considerably shorter than in cases of carcinoma.

Treatment.—In inoperable cases temporary cessations and ameliorations are sometimes effected by treatment with arsenic; and cures have been reported from the mixed toxin treatment as employed by Dr. Coley, of the New York Cancer Hospital—the toxins, *Streptococcus erysipellatis* and *Bacillus prodigiosus* injected hypodermically in doses of $\frac{1}{2}$ to $\frac{1}{4}$ minim and upward.

BENIGN NEOPLASMS OF THE INTESTINE.

The benign growths that develop in the intestine are *adenomata*, *polypi*, *lipomata* and *myomata*. After the diagnosis has been established the treatment must be exclusively surgical.

CHAPTER XLIX.

APPENDICITIS.

APPENDICULAR INFLAMMATION — CIRCUMSCRIBED PERITONITIS —
PERITYPHLITIS — PARATYPHLITIS — SCOLECROIDITIS — SCOLECITIS.

ACUTE APPENDICITIS.

APPENDICITIS is an inflammation of the appendix vermiformis. Acute appendicitis is an infectious disease, induced by bacteria which enter either from the intestinal canal (enterogenous route) or through the circulation (hematogenous route). The infection may rapidly subside, or it may attack the entire appendix and involve the peritoneum. The appendix is exposed to all the diseases of the intestine, particularly catarrhal affections; the enterogenous transmission of the pathogenic agents is easy. When, in consequence of catarrhal conditions, the mucous membrane of the appendix becomes swollen, communication with the cecum is obstructed, the secretions stagnate behind the obstruction, and the imprisoned bacteria set up an inflammatory process. A similar condition may result from fecal concretions (enteroliths), from angular bending of the appendix, from stenoses in the appendicular lumen, from abnormal length or position of the appendix, from adhesions, or from swelling of the lymphatic elements of the appendix. Appendicitis may develop through the hematogenous route from infectious diseases and in connection with osteomyelitis, enteric fever, dysentery, tonsillitis, furuncles, influenza, or oral sepsis (see page 290). It is often secondary to tonsillar or dental infection. The character of the bacteria, which are usually streptococci, is altered by their growth in the mouth, so that they have an affinity for appendicular tissue. The streptococci which inhabit the gastro-intestinal tract do not possess this affinity.

Pinworms are prone to invade the appendix from their habitat in the cecum and may induce appendicitis (see page 803). Accumulating in the appendix in such numbers that they block the lumen, violent expulsive contractions result, furnishing the clinical picture of appendicular colic. Relief follows a partial emptying of the appendix by means of these contractions, and the colicky attack is not repeated until the appendix is again distended. Worms of various kinds have on many occasions been found within the appendix. Cases are reported where worms were found fixed to the mucous membrane, and even embedded in the parietes.

Attention was long since called to the relationship between appendicitis and visceral ptosis. Glénard has discussed gastro-enteroptosis in this connection (see Chapter XXX). Edebohl's believes that from 80 to 90 per cent. of women who have movable right kidney have chronic appendicitis also. This frequency renders chronic appendicitis one of the chief symptoms of movable kidney; and in view of the protracted suffering and serious impairment of health which it entails, and the dangerous possibilities of concurrent acute attacks, it may be considered the most important complication of movable right kidney.

Two varieties of the disease are recognized—simple acute appendicitis and destructive appendicitis.

Simple acute appendicitis is nearly always a catarrhal affection of the mucous membrane of the appendix, spreading to the adjacent intestine. As a rule the peritoneum does not participate, but sometimes the serous covering is slightly affected. In this, the lightest form of appendicitis, the mucous membrane and the follicles are swollen and covered with mucus and secretions, showing small hemorrhages and erosions. When the secretions escape into the cecum the process may recede; but when this does not take place there may develop a phlegmonous inflammation of the appendix with deep ulceration and moderately intense fibrinous peritonitis. This stage may undergo resolution with retrogression of the inflammation and the establishment of an open lumen. The alterations in the appendix after the inflammation may be very slight, such as small cicatrices and thickening of the walls, or obstructions and strictures may develop which will later cause relapses. When the phlegmonous process is not arrested, *destructive appendicitis* is established, with ulceration, empyema and gangrene, leading to perforation. Perforation is succeeded either by an appendiceal abscess or by the development of a general purulent peritonitis, depending upon the protective adhesions formed between the peritoneum and the intestine.

The cecum is a region in which intestinal stasis, within certain limits, is normal, and, because of this, the lymphadenoid tissue is abundant. In the vermiform appendix the same condition exists but in an exaggerated form, and here the lymphoid tissue is the chief characteristic. When there is a swelling of the mucous membrane (edema, catarrhal inflammation) of the cecum, the normal process of emptying the appendix is retarded, due to the narrowing of the aperture into the cecum, and abnormal appendicular stasis occurs. Under such circumstances the lymphoid tissue increases, the nodes swell, and, if the organisms taken up by them are pus-producers, suppurative appendicitis results.

Symptoms.—In cases of simple acute appendicitis the symptoms rapidly subside in conformity to the pathologic course described above, and for this reason the entire process may disappear in

two or three days. But when the anatomic alterations in the appendix are progressive, the clinical symptoms likewise increase in gravity. The greater the pathologic change, the more the symptoms of destructive appendicitis and of general septic infection become evident. These progressive symptoms include a feeling of intense malaise, a greatly disturbed general condition, an anxious, drawn facial expression, great weakness, frequent vomiting, and fever. The disturbance in the general condition depends especially upon the virulence of the infective agents. At the same time the local symptoms become aggravated in a corresponding manner. The pains may be very severe, and McBurney's point so sensitive that the lightest touch is almost intolerable.

In most of the cases there is a marked degree of cutaneous hyperesthesia. In acute inflammation of the appendix absence of pain is no indication that the most serious mischief is not going on; the initial pain of acute inflammation of the appendix, which is so commonly referred to the umbilicus, is due to the dragging upon the mesocecum or the mesoappendix. The cessation of this pain without improvement in the other symptoms is due to cessation of peristalsis, a result of the spreading of the inflammation. Severe pain is of great importance, as it implies either wide extent or great severity of inflammation. Sudden cessation of the pain without corresponding general improvement suggests that the appendix has become gangrenous.

When an abscess has fully formed, a tumor can be made out above Poupart's ligament; the tumor can be palpated from the rectum or the vagina, and this procedure should never be omitted. Increased muscular rigidity (*défense musculaire*) over the region of the cecum should be particularly noted as indicating the involvement of the peritoneum. There may also be increased subjective and objective tenderness extending into the left side of the abdomen and into the lumbar region.

If the appendix lies retroceally, an abscess may develop in the lumbar region, which sometimes reaches up to the lower pole of the kidney or the hilus of the liver. This condition can often be recognized by protrusion or resistance in the loin just above the iliac crest. This spot is also the main seat of pain upon pressure.

There is an increased leukocytosis, accompanied by high fever and rapid pulse. In grave cases watery diarrhea is frequently present. The absence of a perityphlitic tumor when there are severe general symptoms points to the probability that the pus has not become walled off. When general peritonitis is well established, it shows its own characteristic symptoms in gaseous distention, paralysis of the intestine, diffuse sensitiveness to pressure upon the abdomen, and collapse.

Diagnosis.—The commencement of simple acute appendicitis is nearly always rather sudden, marked by more or less pain in the

region of the appendix, nausea, and vomiting. Chills are often present. The bowels are usually constipated, but sometimes loose. There is fever, with acceleration of the pulse. Pain at McBurney's point is elicited upon pressure. This is important, and a detail of the location, as stated by McBurney, is herewith given:

"Whatever may be the position of the healthy appendix as found in the dead-house (and I am well aware that its position when inflamed varies greatly), I have found in all my operations that it lay, either thickened, shortened or adherent, very close to its attachment to the cecum.

"This, of course, must in the early stages of the disease determine the seat of greatest pain on pressure, and I believe that in every case the seat of greatest pain, determined by the pressure of one finger, has been exactly between an inch and a half and two inches from the anterior superior spinous process of the ilium on a straight line drawn from that process to the umbilicus."

While exerting pressure over McBurney's point, the elevation of the right leg of the patient contracts the psoas muscle and forces the painful appendix against the finger. For this valuable sign, which often assists in a differential diagnosis, we are indebted to S. J. Meltzer.

Blumberg's sign, which consists of a short acute pain felt by the patient when the examiner's finger is pressed over McBurney's point and lifted up suddenly, is usually present. This sign occurs in all forms of acute peritoneal inflammation.

Blaisdell emphasizes the importance of the turning test in the diagnosis of acute appendicitis. He found that in cases of suspected acute appendicitis the patient will nearly always be found lying either on the back or on the right side, pain being experienced when he turns on the left side. This holds good in 90 per cent. of all cases. Even if other symptoms are absent Blaisdell believes that turning on the left side is a positive test. In making the test the abdomen must not be allowed to touch the bed, lest the support thus given to the viscera prevent the additional pain which results from the dragging on the sensitive appendix.

In the male, traction on the right spermatic cord induces severe pain near the internal abdominal ring, due to irritation of the parietal peritoneum. The cord is to be grasped between the thumb and index finger, above the testicle, care being exercised not to press unnecessarily on the latter.

On the whole the diagnosis of appendicitis is not difficult. In considering the differential diagnosis, attention should be given to diseases of the female organs, to enteric fever, to diseases of the gall bladder, and to the pseudo-appendicitis of hysteria. A movable cecum is occasionally found as a complication of appendicitis. This condition, known as *cecum mobile*, which in itself may give pain similar to that of appendicitis, is caused by a congenital

malformation of the mesocolon of the cecum. For some distance the mesentery is not attached to the parietal peritoneum, and this permits freedom of movement and displacement. If the patient stands, the pain is more severe, but it is quickly relieved by lying on the back or on the right side. Chronic constipation that does not respond to laxatives is suggestive of cecum mobile. The Roentgen ray shows that the bismuth remains in the cecum for two or three days (see page 147).

Occasionally the lymph nodes of the mesentery in the cecal region will become infected, giving the clinical symptoms of an acute appendicitis. The source of the infection is usually within the appendix, so that the treatment of right inguinal mesenteric lymphadenitis is the surgical removal of the appendix.

Absence of fever, multiplicity of the painful spots, cutaneous hyperesthesia, the contrast that exists between the state of the general health (which is good) and the local symptoms (which are very pronounced) these together would justify rejecting the diagnosis of appendicular lesion. If there is merely abdominal neuralgia we shall discover the classical pain spots, and treatment will clear up any lingering doubt.

Great difficulty is occasionally experienced in forming a clear picture of the anatomic conditions at every stage of appendicitis. For it may happen in some cases that notwithstanding the existence of a most severe destructive appendicitis the symptoms present are slight, or *vice versa*. The typical appendicular symptoms may sometimes become localized in the left iliac fossa, especially when the appendix is very long or when it is transposed toward the left. This condition must not be mistaken for acute perisigmoiditis and diverticulitis (see page 785), which have all the symptoms of an acute left-sided appendicitis.

CHRONIC APPENDICITIS.

Acute appendicitis can only be considered entirely cured when the pathologic changes in the appendix have disappeared and the lumen has not been left stenotic. Stricture is a very frequent sequela, and offers a rational explanation of the great frequency of relapses.

Symptoms.—The course of chronic appendicitis may be such that the primary acute attack is followed by numerous recurrences. The latter may vary greatly in intensity, from the slightest colicky pains of but a few hours' duration (caused by the appendix attempting to push its contents into the cecum) to the most severe attacks. As a rule the subsequent pains are less severe than the first acute seizure. The course may be such that no acute attack has been noted at all, but the patients constantly complain of pains in the region of the appendix, particularly during bodily movements.

on stooping, lifting, or while at stool. These cases of appendicitis running an insidious course without acute attacks are usually benign, although they inconvenience the patients greatly. Another variety is that in which painful sensations remain in the cecal region after a single acute attack, without an acute recurrence at any time. The name "appendicitis larvata" has been applied to a concealed chronic form with symptoms pointing to disease of the gastro-intestinal tract and frequently mistaken for nervous dyspepsia or intestinal catarrh (see page 427).

Ewald was the first to throw light upon this subject. He presented a number of cases which he termed collectively appendicitis larvata, or masked appendicitis (see page 419). The cases had many of the obscure dyspeptic symptoms which are sometimes classed as belonging to nervous dyspepsia. None of the patients gave a history of appendicitis, yet it was possible by careful palpation to diagnose chronic, subjectively unnoticed, appendicitis. Appendicitis larvata presents no characteristic symptoms, and the diagnosis can be made only by excluding diseases of the gall bladder, stomach, and pelvis, and by means of the Roentgen ray (see Chapter V) and the positive objective findings.

Diagnosis.—Rovsing describes a sign that is important as a differential diagnostic point. It consists of pain in the appendiceal region following pressure upon the left side. This symptom is not met with in kidney diseases, ureteritis, stones in the ureter, or salpingitis; but only in diseases of the appendix and cecum. The procedure consists in placing the fingers of the left hand flatly upon the abdomen and pressing slowly down with the right hand along the brim of the pelvis into the left iliac fossa, so that the coils of the ileum are pushed inward out of the way and the fingers press the descending colon firmly against the posterior abdominal wall. The fingers that firmly compress the descending colon are now moved slowly upward toward the splenic flexure. By this manipulation the contents of the gut are put under high enough tension to effect a back pressure upon the cecum. By this pressure a severe lightning-like pain is produced in the ileocecal region. Chase has applied the term "cecal distention test" to this procedure; he believes that a gaseous compression wave will travel across the transverse and down the ascending colon and on arriving at the cecum will produce cecal distention, yielding a typical sharp pain in the right iliac fossa, if inflammation of the cecum or appendix be present.

Rutkevich recommends exploration of the cecum by abduction. The flexed fingers of the right hand are worked between the external wall of the cecum and the abdominal wall; then by extending the fingers the examiner tries to push the cecum toward the median line of the abdomen. In case of chronic appendicitis this will cause pain.

Bastedo recommends the dilatation test for chronic appendicitis. To make this test, a colon tube is passed a few inches into the rectum, and air is injected by means of an atomizer bulb. If, as the colon distends, pain and tenderness to finger-point pressure become apparent at McBurney's point, there is chronic appendicitis.

Morris has found by pressure a painful point on McBurney's line, an inch and a half from the umbilicus, which is especially sensitive in chronic appendicitis. The sensitiveness and pain appear to be due to an irritation of the right sympathetic. When the irritation originates from a pelvic, tubal, uterine or hemorrhoidal affection, one observes two corresponding painful points, one on each side of the umbilicus.

*Aaron's Sign.*¹—This consists of a referred pain or distress in the epigastrium, left hypochondrium, umbilical, left inguinal or precordial region from continuous firm pressure over the appendix. I have found this sign exceedingly valuable in deciding when and when not to recommend operation for chronic appendicitis. The test has been repeatedly confirmed by operation and it is usually found in such cases that the appendix is distorted, adherent, or contracted. The referred pain in these cases is due to spasm of the pylorus or duodenum.²

Transitional leukocytosis or an increase in large mononuclears and in transitional leukocytes is, according to Friedman, suggestive of chronic appendicitis.

Treatment of Acute Appendicitis. The ideal treatment of acute appendicitis is operation within the first twenty-four to thirty-six hours after the onset of the disease. This represents today the best therapeutic endeavor for the treatment of acute appendicitis. It radically removes the disease and cures the patient in the shortest possible time.

Guerry reports a mortality rate of 0.3 per cent. in a series of 545 appendicular operations. He says the third- and fourth-day acute cases are the ones that die. There is a definite and unmistakable tendency toward localization in cases of appendicitis complicated by the presence of pus. In this series of 545 there were 213 of this variety—a very large proportion. Of these 213 cases, 68 patients were seen for the first time on the third or fourth day of the disease. The pulse in most cases was 135, temperature 104° F.; vomiting, distention, pinched features and more or less delirium were also present. Such cases as these are the ones in which operation is attended with mortality. None of these patients were operated upon immediately; all were treated according to the Ochsner method; they were tided through the period of great

¹ Charles D. Aaron, A Sign Indicative of Chronic Appendicitis, *Journal of the American Medical Association*, February 1, 1913.

² Charles D. Aaron, Chronic Appendicitis, Pylorospasm and Duodenal Ulcer, *Journal of the American Medical Association*, May 29, 1913, p. 1843.

danger, and several days later were safely operated upon for localized appendicular abscess.

The Ochsner method has been greatly misunderstood by the profession; when properly applied it is a life-saving measure and entitled to the highest consideration. It was never intended that it should take the place of operation, but that it should permit the selection of a safe time for operation. In cases in which the patients or their friends absolutely refuse an operation the pharynx should be promptly cocaineized, and the patient's stomach irrigated (through the stomach tube) with warm normal salt solution; then food and cathartics by mouth should be forbidden until the patient has been normal for four days, nutrition in the meantime being effected by means of enemata every three or four hours, consisting of some concentrated liquid food dissolved in three ounces of normal salt solution.

Ochsner's method of treatment to reduce mortality involves the following suggestions: In chronic recurrent appendicitis, operation during the interval between attacks. In acute appendicitis, operation as soon as the diagnosis is made, provided the infectious material is still confined to the appendix, and a competent surgeon is available. In acute appendicitis and in peritonitis, without regard to the treatment contemplated, food and cathartics by mouth to be absolutely prohibited and large enemata never given. To relieve nausea, vomiting, or gaseous distention of the abdomen—gastric lavage. When the infection has extended beyond the appendix, watch and sustain the patient until operative intervention is safe. At the beginning, let the thirst be quenched by rinsing the mouth with cold water and by the use of small enemata. Later small sips of very hot water frequently repeated may be allowed, and still later small sips of cold water.

All these cases are greatly benefited by normal salt solution, given by the Murphy drip (see page 239). It should be constantly borne in mind that even the slightest amount of liquid food of any kind by mouth may give rise to dangerous peristalsis and may change a harmless (circumscribed) into a dangerous (diffuse) peritonitis.

This treatment is always indicated, without regard to whether an immediate operation is or is not contemplated. The physician should be on his guard against erroneous deductions from the very rapid improvement of apparently serious cases; there may be, after all, gangrene or perforative appendicitis, which would render premature feeding fatal.

Surgeons and physicians do not, however, agree in respect to the question of whether the lighter and milder cases should always be operated upon immediately, or whether it is proper to treat them expectantly pending the development of serious symptoms. Many physicians maintain that in cases taking the lighter course the

early operation is not imperative, basing this view on the mortality statistics.

An acute attack of simple appendicitis running a mild course need not be operated upon early. The modern experienced physician should be capable of picturing to himself clearly the pathologic conditions present at each stage of the disease while utilizing all the diagnostic adjuvants at his disposal. Thus he will not fail to notice the progress of the inflammatory process. If the diagnostic signs point to the existence of a simple acute catarrhal appendicitis, internal treatment may be adopted. When about to decide the question whether the early or the late operation should be selected, it is of course necessary to pay attention to the local and particularly to the general symptoms. When a practitioner is called to a case of appendicitis he will advocate early operation when the general condition is bad. Grave disturbances in the general condition always indicate high grades of virulency of the infection. A case, therefore, cannot be considered a light one when it exhibits great prostration, lassitude, frequent emesis, anxious facial expression, and slight cyanosis. These features of the case are generally accompanied by grave local symptoms pointing to the conclusion that the peritoneal cavity has become infected, viz.: marked spontaneous pains, increased sensitiveness to pressure not only in the cecal region but also toward the left and as far as the lumbar region, and the very characteristic board-like hardness of the muscles (*défense musculaire*). These three cardinal symptoms necessitate early operation, even though there be no severe general symptoms.

The inflammatory exudate may be so slight during the first twenty-four hours that it is impossible to feel any exudative tumor. Early operation is indicated, however, when the pulse, the temperature and the leukocytosis show marked increase (temperature 101° F., pulse 115, leukocytes 20,000). Thus when the general aspect of the patient is unfavorable, when the pulse, temperature and leukocytosis are equally greatly raised, and when there are symptoms of peritoneal infection, it may be taken for granted that the simple acute appendicitis is passing on to the more serious form, and operation should not be delayed one moment.

When, on the contrary, the general aspect of the patient is satisfactory, the pulse, temperature and leukocytosis not high (temperature 99° F., pulse 95, leukocytes 14,000), and when the local symptoms are moderate, it may be assumed that the further development of the case will be benign, and under such circumstances conservatism and medicinal treatment are justifiable and operation may be deferred.

In any case in which the leukocytes reach or exceed 20,000 or 30,000, the patient should be given over to the surgeon. The characteristic feature of abscess formation is that from the beginning

—or at least from the second day on—the number of leukocytes rises rapidly and maintains itself, with but slight variations, at the level reached, or goes even higher. After operation the number usually falls immediately, although in rare cases it may rise higher than it was before. In the diagnosis of abscess formation, observation of the behavior of the leukocytes far exceeds the temperature in value. If there is a sudden subsidence of pain, and other symptoms show that the inflammation has not aborted, gangrene may be present and operation should be immediately done. It has been observed that a high absolute leukocyte count (30,000) with a high polynuclear count (95 per cent.) suggests a good prognosis. A low absolute count (7000) with a moderately high polynuclear indicates a bad prognosis.

The internal medical treatment of simple acute appendicitis should be directed from the very first with a view to the securing of absolute rest, both of the patient and of the intestinal tract. For this purpose the patient is ordered to bed, which must not be left even for micturition or defecation. The bathing of the patient must also be done in bed. An ice-bag of moderate weight should be placed over the cecal region and kept there day and night. When sleep during the night is interfered with by the ice-bag, it may be omitted and a Priessnitz bandage substituted. When the pains are marked, the ice-bag should be suspended on a bed hoop in such a manner as just to touch the cecal region while hanging. Opium, which formerly was regularly given, may usually be omitted in light cases. It must not be forgotten that opium may mask the actual condition; it should therefore be given only in case of very severe pain, and then only in the smallest effective doses.

Recently the administration of purgatives has again been advocated, a practice which was once considered strictly contra-indicated in acute appendicitis. Sonnenburg, who has made a special study of appendicitis, employs castor oil in the treatment—on the ground that evacuation of the bowel, and particularly of the appendix, assists in removing stagnating infectious materials. It is not certain that the peristaltic intestinal movements induced by the castor oil are capable of emptying the appendix. In simple catarrhal cases the evacuation of the bowel by castor oil is accompanied by the disappearance of the sensitiveness to pressure and by a feeling of relief to the patient, promoting more rapid retrogression of the disease. Castor oil has also the advantage of shedding light on the prognosis and on the course of the disease; for, should it prove ineffectual, the attack not declining at all, and no movement of the bowel occurring after several hours, it may be gathered from these circumstances that an evacuation of the appendix has not taken place, being probably impossible because of the prevailing pathologic conditions. Operation should then be undertaken at once. The treatment with castor oil is permissible only when the

patient is under constant surgical observation in a private sanitarium or in a hospital. It is absolutely unsuitable in the patient's home.

Irrigation of the bowel, and enemata, are not to be employed in the acute stage, although they are recommended by some authors. As the patients partake of but little food during the attack, it is sufficient to begin about the sixth day to give very small oil enemata, or water injections with oil and soap, repeating daily. Glycerin enemata are contra-indicated because of the fact that they induce marked peristaltic movements.

With respect to diet during the acute attack: The patient should take no food during the first twenty-four hours or even longer. The thirst should be quenched by small pieces of cracked ice and by the moistening of the mouth with water. During the next few days iced milk should be given in sips, and then small amounts of gruel and some beef tea. This, after the attack passes off, is to be followed by beef tea with egg, milk, soup, and tea; and later, gradually, finely chopped tender meat, mashed potatoes, vegetables and apple sauce may be allowed. After the first natural bowel movement the diet may become somewhat more free. Great care should be exercised with the diet for a considerable length of time after the attack; all food should be cut up minutely, and all strong, irritating articles of food should be avoided. In the event of constipation supervening, it should be counteracted by a chemically purgative diet. (See Chapter VII on Diet.) Fatiguing bodily activity, exercise, golf, sports and abdominal massage are to be forbidden for a long time and in some cases for years.

After the acute attack, it is the best practice to remove the appendix at once, or, if this is impracticable, to allow an interval of about ten days to elapse. The operation should take place when all the inflammatory symptoms have subsided and when all adhesions which may have formed have become firm. At this time it is neither difficult nor serious; the wound can be closed and allowed to heal by first intention, and the mortality in cases of simple appendicitis does not exceed 0.6 to 0.9 per cent. It must be insisted upon that appendectomy is the only certain prophylaxis which will positively exclude recurrences. Internal remedies are absolutely useless in this respect.

There is a large class of cases which must be treated medically, because for one reason or another it is impracticable to operate early. The patient may be in the country, and operation may be inadvisable because of old age or the presence of lung or heart disease or diabetes. There are also instances in which the patient refuses to permit an operation, and those obscure cases in which it cannot be decided positively whether the disease is appendicitis, cholelithiasis, pyosalpinx, typhoid fever, or hysteria. In such cases the usual treatment, as described above, is to be followed out. When more serious symptoms supervene, opium should be

given, because by inhibiting peristalsis it prevents traction of the intestine upon the inflamed peritoneum, thus counteracting any tendency toward the development of peritoneal infection. Local applications of ice are to be persistently maintained, but purgatives positively prohibited. Absolute bodily rest is to be insisted upon, that perforation from abscesses possibly present may be prevented. The diet should be as already described.

Under the conservative treatment severe cases sometimes recover, but, generally speaking, it is impossible to prevent the development of destructive appendicitis by internal medication. In case of recovery, subsequent operation must be performed. Without such operation these cases of appendiceal abscess show a high mortality, as against 0.6 to 0.9 per cent. after early operation.

When operation is delayed until the third day, the conditions are, as a rule, much less favorable than those of early operation. It is very rare indeed that the process at that time continues to be as suitable for surgical treatment as on the first two days; moreover, these cases present a bad outlook either with or without an operation. When encapsulation has not taken place the process usually becomes progressive. If a circumscribed abscess has been formed, there is no immediate danger of death; the advice of the surgeon, therefore, is not to interfere with this process of encapsulation by operative procedure; operation, however, offers relatively good chances if the abscess can be evacuated without opening the peritoneal cavity. The intestine may perforate spontaneously by rupture of the abscess into it, and by this means a cure be effected by nature.

If signs of peritonitis develop, continuous saline infusions into the colon by the Murphy drip should be given. This method of administering saline solution is claimed to be of such great value in diminishing toxemia and in saving life that a detailed description of it is given on page 239. Under this treatment the blood-pressure is restored to normal, thirst is quenched, and septic products are more readily excreted.

Morphin or opium should be administered in sufficient quantity to stop peristalsis. Stockton¹ does not advocate the opium treatment to the displacement of surgery in general septic peritonitis, but believes, with Alonzo Clark, that its greatest field of usefulness is in those comparatively mild cases in which the inflammation has not yet become general. Clark taught that as soon as the diagnosis was made the largest safe dose of opium should be administered. He aimed to use very large doses, with long intervals between, yet never to allow the patient to emerge from the sedative effects of the drug. In general peritonitis the amount of opium and morphin that can be taken safely is remarkable. The aim of the treat-

¹ Charles G. Stockton, *The Opium Treatment of Peritonitis*, *Buffalo Medical Journal*, February, 1908, p. 373.

ment is to keep the patient as quiet as possible and yet within the borderland of safety. To one experienced with the drug in this disease, there is little danger of overuse. As the inflammation subsides, the tolerance for opium decreases and the dosage is lessened, and very soon it is unnecessary to administer any anodyne whatever.

Stockton teaches that the proper treatment of oncoming septic peritonitis is immediate operation, but that when the operation is delayed and the inflammation is becoming general it is advisable to use the full opium treatment until such time as the suppuration is localized and drainage effected without molesting the greater part of the abdominal cavity.

Treatment of Chronic Appendicitis. Removal of the appendix after an acute attack will, of course, prevent recurrence and the more insidious advances of chronic appendicitis. No internal medicine offers such security. There may be small ulcers or abscesses in the appendix in apparently cured cases, which at any moment may excite an acute attack. When operation is not performed, the patients are compelled to alter their entire mode of living, for the sake of prophylaxis, into a regimen of rest and observance of the strictest dietary precautions. The great majority of people are incapable of persevering and are unwilling to submit to such restrictions; many of them, of their own initiative, demand an operation. The chances without operation are even worse in the case of children, because of their natural restlessness.

When an operation is refused, it is necessary to demand of the patient that he avoid bodily exertions, give up gymnastics and hard bodily labor, take most particular pains not to commit any errors in diet, and avoid any external irritation in the region of the cecum. Particular attention must be given to regularity of the alvine discharges.

The food should be carefully subdivided and heavy residual matter avoided. When constipation is present the diet should be of such a nature as to act chemically as an evacuant (see page 185). Cascara-agar should be employed as an adjunct to the diet. Rectal injections of oil may also be employed (see page 224). Liquid petrolatum (see page 664) is a soothing lubricant to the mucous membrane and prevents irritation by scybala; it is antiseptic, emollient and laxative, and can be given in tablespoonful doses three or more times a day. Menthol or thymol may be added to the dose when desired.

In these non-operative cases, systematic drinking cures may be undertaken at Saratoga, Carlsbad, Marienbad, Kissingen, or Wiesbaden. Favorable effects are also frequently obtained from mud baths (Mudlavin, Indiana), also from local applications of mud. The absorption of exudates of long standing is often promoted by means of these baths and applications. The use of the

hot-water bag, hot-water stupes, oil and turpentine, linseed or mashed-potato poultices, Priessnitz bandages, and thermophores has the same purpose and similar effects. Massage and purgatives are contra-indicated.

When these measures are observed for a long time and with strict regularity it is occasionally possible to relieve the condition and remove the danger of relapse, although this result cannot be assured. Such a very guarded method of living cannot be maintained by all (*e. g.*, workingmen), and it may become necessary after all to operate.

Bacterial vaccines have been used in the treatment of appendicitis with occasional success. Colon bacillus, streptococcus, staphylococcus, pneumococcus and pyocyanus infections are the bacterial causes of peritonitis in appendicitis, and immunization against these organisms in appendicitis is unquestionably of benefit whether the case is to have an operation or not. A mixed polyvalent stock vaccine, consisting of the above-named organisms, should be given. Specific agents which have the power to neutralize toxic microbial products, destroy the microbes, or dissolve foreign protein substances, are called antibodies. Substances which, introduced into the blood, lead to the formation of antibodies, are antigens. The modified bacterial derivatives prepared according to the process of Schafer consist mainly of antigens. The trade name "phylacogen" has been given to these products. Phylacogens are sterile aqueous solutions of metabolic substances, or derivatives, generated by bacteria grown in artificial media. Pathogenic bacteria, such as *Staphylococcus albus* and *aureus*, *Bacillus pyocyanus*, *Diplococcus pneumoniae*, *Bacillus coli communis*, *Streptococcus hemolyticus* and *viridans*, *Micrococcus catarrhalis*, etc., are employed in the production of these so-called phylacogens, or "phylaxin developers." The mixed-infection phylacogen has been used with good effect in some cases of appendicitis.

CHAPTER L.

NERVOUS DISEASES OF THE INTESTINE.

ENTEROSPASM; TORMINA INTESTINORUM NERVOSA; PARESIS; ENTERALGIA.

THE coördination of vagus and sympathetic nervous impulses regulates the digestive functions. The stomach and intestine possess ganglionic cells, situated in the muscles and glands, which have the power of allowing the organs to function in an independent manner. There are fibers from the medulla which intermingle with the sympathetic and ganglionic cells so that these organs are also under the influence of the central nervous system. In the normal condition the innervation induces a continuous tonus which oscillates between certain limits. The fibers of the sympathetic, or the splanchnics, check or retard the activity of the digestive organs, especially with regard to secretions and motility. The fibers of the vagus send impulses inducing contraction and tonicity of the muscles of the digestive organs. Every increase of irritation of the vagus increases this effect on the activity of the muscles of the intestinal canal. Excessive irritation, or vagotonia, brings about an increase in the secretion of juices, and spastic contractions of different sections of the intestine (see page 388).

ENTEROSPASM.

Symptoms.—The symptoms of enterospasm consist of painful contractions of the intestine, followed by marked intestinal peristalsis accompanied by borborygmi. When a wave of peristalsis passes down on a segment of intestine that is already spastically contracted, the result is cramp or colic. During the height of the painful seizure the necessity for defecation becomes urgent, and the discharges, expelled with pain and tenesmus, consist of small fecal fragments in the form of narrow cylinders or balls. The attacks occur spasmodically, and may occlude the part of the intestine involved. Mental and nervous conditions may give rise to these attacks. The diagnosis is not difficult, in view of this typical course and the absence of any organic disease.

Treatment. The therapeutic measures must be particularly directed toward the general nervous system. Physical and hydropathic procedures are valuable. A hot bath is grateful and gives quick relief. Galvanism applied to the intestine, with one flat

electrode over the abdomen and the other in the rectum, is useful. Both prophylactically and during the attack, warm applications to the abdomen should be made. Carminative teas will frequently relieve the spasm (see page 658). Anodynes, such as opium, belladonna, morphin, hyoscyamus, and the bromids, are to be employed. The nitrites, by inhibiting peristalsis, relieve the cramp or colic. Suppositories of the anodynes frequently give quick relief. Papaverin and benzyl benzoate are also efficacious (see page 276). Chloral hydrate dissolved in warm water can be introduced into the rectum. Hot oil enemata are valuable. Chronic constipation should be appropriately treated (see page 659).

TORMINA INTESTINORUM NERVOSA.

Peristaltic restlessness was first described by Kussmaul. It consists in the development of intense peristaltic motions, visible through the abdominal walls. It occurs in nervous patients, especially under the influence of exciting emotions. During the attack the patients experience a feeling of distention and contraction in the abdomen, associated with pain and loud intestinal noises. These symptoms are not dependent on the ingestion of food. The active peristaltic movements are quite apparent on inspection of the abdomen, especially when the abdominal walls are thin. The small intestine is usually the part most prominently involved, but the colon may participate. The attacks often persist for a long time, and may vary considerably in their intensity. When making a diagnosis the possibility of stricture of the intestine must be borne in mind (see page 755).

Treatment. The treatment is directed chiefly toward the strengthening of the entire nervous system. All the adjuvants of general physical therapeutics are utilized for this purpose. Change of climate and sojourn in mountainous regions are occasionally quite beneficial. The application of the faradic current to the abdomen, stomach and rectum is sometimes successful. Rest cures and hyperalimentation (see page 569) are frequently of great value. The bromids, opium, belladonna, codein, chloral hydrate and strychnin may be given.

		Gm., or Cc.	
R	Tincturæ belladonnæ,		
	Tincturæ nucis vomicæ	aa 10 0	3 iiss
Misce			
Sig—	Fifteen drops three or four times daily		

PARESIS OF THE INTESTINE.

Paresis of the bowel is a functional relaxation of the intestinal muscle fibers, and is found in chronic constipation and in various organic intestinal affections; also as a result of the continued use of opium, morphin, or belladonna; and from neuroses, psychoses, and

gallstones are to be borne in mind. Hernia of the linea alba must not be overlooked.

Treatment.—The treatment should be directed toward the original cause. Symptomatic indications are offered for the application of heat, hot enemata, galvanization, and anodynes.

Chloral hydrate in combination with morphin and belladonna is useful:

	Gm. or Ce.	
R̄—Morphini hydrochloridi	06	gr. j
Chloralis hydratis	12 0	3ij
Syrupi aurantii, Aque	3ā 30 0	3j

Misce.

Sig.—One or two teaspoonfuls every hour during the attack.

For the relief of severe pain:

	Gm. or Co.	
R̄—Codeinæ phosphatis	1 0	gr. xv
Acidi acetylsalicylici	4 0	3j

Misce et ft. caps. no. xvi.

Sig.—One every hour until better.

The treatment is similar to that of gastralgia (see Chapter XVIII).

CHAPTER I.I.

PERISIGMOIDITIS—DIVERTICULITIS—PERIDIVERTICULITIS—SIGMOIDITIS; IDIOPATHIC DILATATION OF THE COLON.

PERISIGMOIDITIS AND DIVERTICULITIS.

Acute Perisigmoiditis.—Acute perisigmoiditis is a disease that runs a course very similar to that of acute appendicitis. It is at present comparatively little known. An endosigmoidal and an exosigmoidal origin of the disease are recognized. The affection commences endosigmoidally by direct continuation of inflammatory processes from the descending colon and the upper portion of the rectum (severe catarrhs, fecal stagnation, stercoral ulcers, colitis ulcerosa, diverticulitis) to the peritoneal coating of the intestine and the adjacent peritoneum. A perisigmoidal exudate, either serous or purulent, may develop. These cases are not very common.

Stagnation of the feces occurs mostly in the lower portion of the descending colon and in the sigmoid flexure, the peculiar anatomy of which virtually invites the retention of fecal material. Inflammatory processes of the sigmoidal mucosa may thus be set up readily, to some extent by mechanical injury from the accumulated excrementitious substances, and to some extent by chemical irritation from the products of secondary decomposition evolved from the impacted feces. Intestinal spasm, atony, or paresis, by occasioning constipation, may be the more remote cause of chronic inflammatory processes of the sigmoid flexure. The great majority of all intestinal affections which are due to insufficient or perverse innervation arise in the sigmoid. Initial intestinal spasm is almost invariably confined to the sigmoid; intestinal atony or paresis, as a rule, starts in the sigmoid; 70 per cent. of all cases of volvulus—due primarily in almost every instance to fecal impaction and induced forced peristalsis, that is, perverse innervation—occur in the sigmoid flexure. Chronic sigmoiditis may be associated with numerous concomitants and complications. Its occurrence, in fact, favors the development of other, especially functional, disturbances of the alimentary canal. Functional disorders of the cecum, for instance, are often due to a chronic pathologic condition of the sigmoid. The synchronous occurrence of cecal derangement and the syndrome of chronic sigmoiditis often presents the clinical picture of chronic appendicitis. Again, we know that in the etiology of appendicitis itself, chronic constipation and consequently sig-

moidal disease) plays an important rôle. Furthermore, perisigmoiditis may be due to the same cause which gives rise to endosigmoiditis, or it may be the direct consequence of the latter, and inflammatory processes in other parts of the peritoneum may be engendered by chronic sigmoiditis or its most frequent immediate precursor, constipation.

Perisigmoiditis of exogenous origin occurs quite often. Inflammation, exudation, and the formation of an abscess may occur in the course of diseases of neighboring organs, as the female genitals, the kidneys, or the ureters, or in a case of psoas abscess. It must also be remembered that an exudate from an acute appendicitis may become localized perisigmoidally.

Diverticula may occur in any part of the small or large bowel. Those in the former are as a rule congenital, and those in the descending and pelvic colon usually acquired. Diverticulitis is due to infection in the walls of one or more diverticula; these walls usually show evidence of chronic inflammation in the mucous and submucous coats, the latter abounding in fat. The diverticulum seldom shows longitudinal or circular muscle fibers, but frequently contains hard, black fecal concretions. Hyperplasia of all adjacent glands occurs, and a large inflammatory mass is caused by outward bacterial invasion. Fecal concretions frequently cause chronic irritation, and produce a secondary peridiverticulitis which may eventuate in abscess formation.

Diverticulitis.—Diverticula of the small intestine are rare, although several cases have recently been reported recognized by the Roentgen ray. They are more often found in the duodenum than in the jejunum or ileum.

Diverticulitis of the sigmoid may be either congenital or acquired. These small hernial protrusions of the coats of the bowel may occur anywhere in the large intestine, but are most common in the sigmoid, ending abruptly at the rectum. Unless they undergo secondary changes they do not give rise to any symptoms (Eisendrath). Multiple diverticula always occur in large numbers; in some instances several hundred of them are distributed over the entire large intestine, notably in the sigmoid flexure and the rectum. Diverticula are most apt to occur in two rows, at either side of the gut. They may, however, develop at the mesenteric attachment and appear between the layers of the mesentery. In some cases they are simply protrusions of the mucous membrane into the appendices epiploicæ, while in others they form definite pouches, either oval or flask-shaped.

The sigmoid loop in which the diverticula are found is frequently filled with fat, and Telling¹ has demonstrated that this fat entirely conceals the hernial protrusions, more especially as these are

¹ W. H. M. Telling. The Clinical Aspects and Importance of Sigmoid Diverticula. *The Practitioner*, March, 1911, p. 14.

mostly into the epiploic appendages. Even when the fat is present only in moderate amount the condition is easily overlooked by any one not familiar with the characteristic appearances. Fig. 110 illustrates a well marked case with the fat removed from the outer aspect of the bowel. Fig. 111 illustrates the inner surface of the same specimen. Fig. 112 illustrates a case in which the fat has been dissected from one half of the bowel, and shows the



FIG. 110 Diverticula of the sigmoid flexure. The fat has been dissected from the outer aspect of the bowel. The pouches are for the most part into the appendices epiploicae. *a*, one of the sacs open. (Telling.)

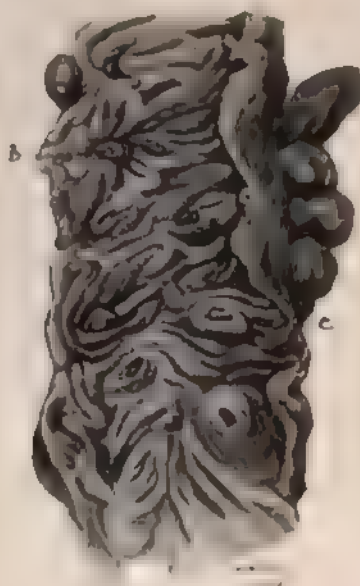


FIG. 111—Diverticula of the sigmoid flexure. Inner surface of the bowel. *b*, a concretion at the orifice of one of the diverticula, *c*, lipped orifice. (Telling.)

pouches which entered the appendices epiploicae. The diverticula usually contain fecal matter or concretions of varying degrees of hardness. Serious results may follow the thinning of the diverticulum wall, the perforating action of the retained concretions, and the presence of microorganisms and their toxins. The most frequent pathologic effect of these diverticula is a chronic proliferative inflammation and the resultant perisigmoiditis. The change

that takes place is of the proliferative type, and a mistaken diagnosis of carcinoma may be easily made (Fig. 113). This proliferative inflammation involves the whole circumference of the bowel for a varying distance (two to eight inches as a rule), and, besides giving rise to the formation of a definite tumor, the inevitable contraction of the new-formed fibrous tissue leads to a slow stenosis of the bowel.



FIG. 112. Diverticula of the sigmoid flexure. The fat has been dissected from one side of the bowel, showing the pouches which entered the appendices caecales. *a*, a single pouch dissected out to show how they are buried in fat and liable to unrecognized unless especially sought for; *b*, longitudinal muscular band; *c*, a diverticulum (Telling).

One of the most important factors in the production of diverticulitis is muscular weakness incident to old age, and the presence of considerable fat in the intestinal wall which favors a pushing out of the mucosa. The most frequent secondary pathologic changes are: (*a*) infection of the general peritoneal cavity without perforation; (*b*) acute or gangrenous inflammation resembling the same pathologic form of appendicitis; *c*) acute perforation or formation of an abscess, or general peritonitis. These pathologic forms greatly resemble corresponding types of appendicitis.

Multiple diverticula in a non-inflammatory condition are only discovered at autopsy, as they present no clinical manifestations. But the feces contained in them may cause inflammation, necrosis, ulceration, and perforation. The latter is usually of small dimensions and becomes encapsuled or adherent. It may, however, lead to slowly progressing ulceration and diffuse peritonitis. Such a condition may be mistaken for appendicitis.

Clinically a distinction is made between acute simple and acute ulcerative perisigmoiditis. In the former group are classed those cases in which there may be found, aside from the slight general symptoms and sensitiveness to pressure, some resistance in the region of the sigmoid flexure, but the contour of the bowel is not concealed; at the same time there are constipation and slight fever. With proper treatment these phenomena gradually disappear within five to eight days. The cases of ulcerative peri-

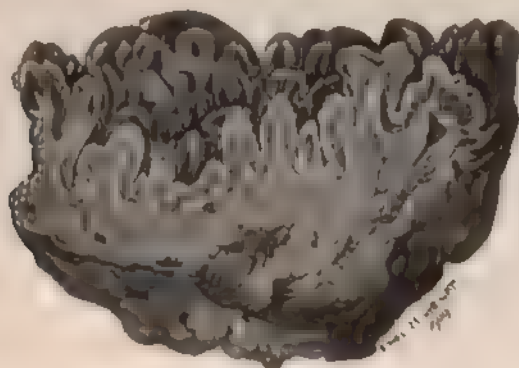


FIG. 112 -- Perisigmoiditis with great thickening of the gut wall, causing stenosis and simulating carcinoma. a, thickening due to fibrosis. (Telling.)

sigmoiditis are those in which the primary symptoms become worse, with high fever and grave general conditions; a large exudate or abscess may develop in the region of the flexure. At this period large abscesses may undergo absorption. Occasionally, however, serious complications arise, such as perforation and peritonitis.

Symptoms.—The clinical symptoms, sudden onset, pains, vomiting and fever, frequently correspond to those of acute appendicitis with symptoms on the left side, and for this reason it is often difficult to establish the correct diagnosis. In cases of diverticulitis we may have gangrene, peritonitis, and death. These diverticula are a series of appendix-like structures and are subject to the same pathologic changes as the vermiform appendix. The appendix itself is only a blind diverticulum of the intestine. The symptoms suggesting an endogenous perisigmoiditis are, a history of coprostases, diarrheas, and the finding of blood and pus in the stools. Rectal and vaginal examinations are never to

be overlooked. The sigmoidoscope, which may frequently clear up the diagnosis of this condition, is not to be used during the acute stage of the disease. In all cases of diverticulitis the diagnosis can be easily made by the use of the Roentgen ray and barium sulphate or bismuth subcarbonate (see page 148 and Plate XXII, Fig. 1).

Treatment.—Prophylactically, careful attention should be directed to coprostasis in the region of the sigmoid flexure, to chronic constipation, and to chronic ulcerative colitis, in order to prevent the development of a perisigmoidal affection. A well developed case is treated similarly to one of appendicitis—absolute rest in bed, ice applied locally, abstinence from food, or slight quantities of fluid or semisolid nutriment, and opium or morphin. In very light cases, in which fecal retention can be demonstrated, very cautious attempts may be made to evacuate the bowel by means of rectal irrigations. The greater percentage of cases of endogenous perisigmoiditis go through to recovery without surgical intervention, a measure which is undertaken more frequently in exogenous perisigmoiditis. Generally speaking, it is permissible to wait longer in perisigmoiditis before operating than in appendicitis.

Many patients suffering from diverticulitis have been greatly relieved after taking the barium or bismuth test meal. Repeated Roentgen-ray examination proves that these metals really have an influence on the multiple diverticula. It has thus been found that barium sulphate or bismuth subcarbonate in 60-Gm. (2-ounce) doses is very valuable in the treatment of cases of diverticulitis. This dose should be taken in the morning on an empty stomach once a week for three or four weeks. These patients do better without cathartics.

Chronic Perisigmoiditis.—After recovery from an acute perisigmoiditis, there may result cicatricial processes of contraction and adhesions, interfering with the passage of feces into the sigmoid flexure. Under such circumstances it may become necessary to resort to surgical intervention.

IDIOPATHIC DILATATION OF THE COLON—HIRSCHSPRUNG'S DISEASE—CONGENITAL MEGACOLON—CONGENITAL DILATATION OF THE COLON.

The term "idiopathic dilatation of the colon," or "Hirschsprung's disease," is applied to a dilatation which usually affects the sigmoid flexure and rarely the entire large intestine. It is accompanied by hypertrophy of the walls of the dilated portion of the gut. No cause can be found for it. Neither a contraction at either end of the dilatation or any other organic change is present. Both the anus and the rectum are normal. The dilatation, as a rule, commences at a point five to seven centimeters (about two inches)

above the rectum, and occasionally higher up. The sigmoid flexure is enormously dilated and presents the appearance of a large loop, which often extends upward as far as the costal arch. In extreme cases the dilatation of the intestinal coil is so enormous that, on opening the abdomen, the other contents of the cavity are entirely concealed. Cases have been described in which the diameter of the gut has measured fifteen to twenty centimeters (five to seven inches) and even more. The descending colon does not usually participate in this dilatation, rarely the transverse portion, and the ascending colon and the cecum almost never. The small intestine is normal in every respect. The walls of the dilated portion of the intestine are markedly thickened and dilated. This thickening is caused partly by hypertrophy of the muscular layers and partly by hypertrophy of the connective tissue.

The etiology of this disease, first fully described by Hirschsprung, and named after him, is, according to our present knowledge, as follows: There is originally an abnormal length and dilatation of the sigmoid flexure (megacolon), developed during fetal life. After birth this abnormality induces disturbances in defecation which lead to muscular hypertrophy and increasing dilatation of the enlarged portion of the bowel. When there are at the same time bendings and angulations of the megacolon, these contribute to the further retardation of intestinal evacuation.

A large proportion of the cases published as Hirschsprung's disease are, in reality, merely the result of kinking of the intestine, usually at the sigmoid flexure, or the result of some primary or secondary anomaly in the mesentery. The assumption of a congenital deformity is sustained by the finding of congenital malformations in other parts of the body (see page 561).

The anatomic conditions in early childhood favor this kinking of the intestine, and any disturbance in the digestive organs is liable to cause trouble from this source. The sigmoid flexure in children kinks easily; and the space in the pelvis, especially in boys, is restricted, as compared with the adult anatomy. Rectal examination is important for differentiation; in cases of dilatation, large amounts of fluid can be introduced without flowing out again. Fluid passes readily upward, but the valve-like closure of the kinked intestine prevents its escape. The Roentgen ray is of great assistance in the diagnosis (Plate XXI, Fig. 1, Chapter V).

The majority of cases of dilatation of the colon are seen in young children, but the condition is occasionally observed in adults. It is a peculiar fact that boys greatly predominate in this affection. One of the very early signs in infantile or congenital cases is irregularity in the evacuation of the bowels. There exists from the beginning an idiosyncrasy in this direction; and the natural constipation is presently followed by great tension and enlargement of the abdomen. The majority of these children die during infancy,

the cause being either intestinal toxemia or acute enteritis. Occasionally, however, the indications of the existence of any disease are so slight during childhood that the children continue to live uneventfully, and the disease induces serious conditions in later years. It is very important to note that the muscle fibers of the dilated intestine soon become hypertrophied. When this has taken place, intestinal evacuation may remain in a fairly satisfactory condition, the disturbances being confined to a slight degree of constipation, with no alarming symptoms. But when the hypertrophy does not develop sufficiently, the children generally succumb during the first year of life. It is probably correct to assume that idiopathic dilatation of the colon is the cause of death in the case of many children who die during the first year of life with symptoms of constipation and without any very clear clinical demonstration of the anatomic abnormality.

Symptoms. The characteristic clinical symptom, as intimated above, is the persistence of constipation, commencing with the birth of the child, and necessitating the aid of purgative medication or enemata. The feces may be soft, but occasionally inside the dilated portion of the intestinal canal there is found a fecal mass of extreme hardness. There may be symptoms of an advanced stage of fecal decomposition. In such conditions purgative drugs are decidedly less effective than enemata. The escape of offensive flatus is frequent. Notwithstanding the good appetite and the ingestion of plenty of food, the state of nutrition becomes impaired in consequence of toxemia incident to the decomposition processes and the constipation. The patients become emaciated. When muscular hypertrophy has become well established, the bowels may be able to expel the accumulated fecal masses from time to time, either spontaneously or with the assistance of appropriate measures. These are the cases in which life can be prolonged into more advanced age. But when the irregularities in the bowel movements are more strongly developed they frequently induce violent colics and symptoms of stenosis. The abdomen continues to be more and more distended, the lower ribs are pushed outward, the lower portion of the abdomen on the left side protrudes considerably, and the veins of the skin over the abdomen become dilated. The liver and the heart dulness, together with the diaphragm, are widely displaced upward, and it is often possible to see the outlines of the enlarged intestinal loop beneath the abdominal wall. As a rule nothing abnormal can be felt per rectum. The feces gradually continue to increase in offensiveness. Very often they contain blood and pus, from ulcerations (distention ulcers) of the mucous membrane. The toxic symptoms become more pronounced in proportion to the difficulty experienced in evacuating the bowels. Finally the patients die, either of this disease or of an intercurrent acute enteritis.

When the patients survive the period of infancy, the prognosis is better. The compensatory muscular hypertrophy is capable of overcoming the fecal stagnation for a considerable length of time, and the growth of the patient in a certain sense repairs the damage. The mesentery ceases to grow, undergoes shortening, and holds the dilated intestinal loop in the pelvic cavity. The mortality, however, even in more advanced age, amounts to 51 per cent.

Treatment.—The internal treatment of idiopathic dilatation of the colon is symptomatic only, and its principal purpose should be to prevent the stagnation of feces. All the measures adapted to the treatment of chronic constipation serve this purpose, but more particularly should copious irrigations be made with the fluids mentioned in Chapter XI; yet these irrigations will not be effective unless the rectal tube is pushed up a considerable distance from the anus. In order to prevent bending of the rectal tube, it is well to guide it with the finger inserted alongside. When the fecal masses are very hard it may become necessary to undertake their direct manual evacuation; in this way enormous quantities of decomposing material are sometimes removed. The distention of the abdominal cavity and the colicky pains may be diminished by the introduction of a rectal tube, thus facilitating the escape of gas. Many authors caution against the administration of purgatives by mouth, especially drastics. These often aggravate the condition, particularly when the sigmoid flexure shows marked convolutions and angular bendings. A few clinicians have observed good effects from the internal administration of senna, rhubarb, and strychnin.

The diet corresponds, on the whole, to that prescribed for cases of spastic constipation (see Chapter VII). The chief requisite is that it yield little residue, thus limiting the amount of fecal material formed.

These measures are to be supplemented by hydrotherapeutic applications, such as Priessnitz bandages, warmth to the abdomen, hot packs, and sitz baths. A regular abdominal massage and the percutaneous or intrarectal application of the faradic current are useful at times (see Chapter X).

On account of the limitations of internal therapeutics in this class of cases, surgical intervention has been undertaken with rather satisfactory results in many instances.

CHAPTER LII.

THE ANIMAL PARASITES OF THE INTESTINE.

TAPEWORMS.

THE tapeworm, a member of the group of cestodes, is found very frequently in man.

Tænia Saginata. The most common form of tapeworm seen in the United States is the *Tænia saginata*, or *mediocanellata* (Fig. 114). It may be identified by its extensive branched uterus and lateral genital pores, and its head with four suction depressions without a corona or hooks (Fig. 115, *a*). Its presence in man is proved by demonstration of its eggs (Figs. 115, *c*) and by the discharge of proglottides (Fig. 115, *b*) or segments of the worm in the feces; the segments are often evacuated independently of the feces. *Tænia saginata*, or the "unarmed tapeworm," enters the intestine through the eating of measly beef.

Tænia Solium. The *Tænia solium* is more rare, and may be identified by its slightly branched uterus with lateral genital pores (Fig. 117, *c*). Its head has four suction facets with a corona of hooks (Figs. 116 and 118). Proof of the presence of this worm in the intestine by the demonstration of ova in the feces is more difficult than in the case of *Tænia saginata*, because the eggs are not deposited so frequently (Fig. 119). Isolated segments and connected pieces of the worm itself are, however, occasionally discharged. *Tænia solium*, or the "armed tapeworm," is acquired by eating measly pork.

Bothriocephalus Latus.—The next variety of tapeworm occurring in man is the *Bothriocephalus latus* (Fig. 120), which is particularly frequent in Holland and the Baltic countries. *Bothriocephalus latus* contains many proglottides, and is recognized by its short broad segments, its small, slightly branched uterus and median genital pores, and the oval head with two lateral suction facets (Figs. 121 and 122). The ova of the *Bothriocephalus latus* may be easily demonstrated in the feces; they are characterized by a lid-like covering at one extremity. *Bothriocephalus latus*, or the "broad Russian tapeworm," is acquired by eating measly fish.

Hymenolepis Nana.—There is another tapeworm that is frequently found in certain parts of the United States. The *Hymenolepis nana*, the dwarf tapeworm (Figs. 123 to 128), is a small tapeworm, less than two inches (5 to 45 mm.) in length. It has four suckers with a corona of hooks on the head, and the genital

pores are lateral. This tapeworm inhabits the ileum, and there may be a single parasite or several thousand present. The detached segments of this worm are so small that they easily escape detection. With the microscope the characteristic eggs can easily be found in the stools. Rats and mice are regular hosts for this worm. The eggs may be carried to the food through the droppings of mice and rats on their visits to the pantry.

There are also *Tænia mada gascariensis*, *Tænia flavopunctata*, *Tænia cucumerina*, *Bothriocephalus cordatus*, and other tapeworms, all of which are rarely met with in the United States.

Mode of Infection.—The mode of infection with these tapeworms is by the original ova entering an intermediate host, developing into cysticerci, and passing, with the flesh of their host (as contaminated meat), into the stomach and intestine of the patient. Thorough boiling of the meat destroys them. The intermediate host for the *Tænia mediocanellata* is beef; for the *Tænia solium*, pork; for the *Bothriocephalus latius*, pike, and frequently salmon. In the case of the *Tænia solium* the [patient himself



FIG. 114. *Tænia saginata*, natural size (Günther).

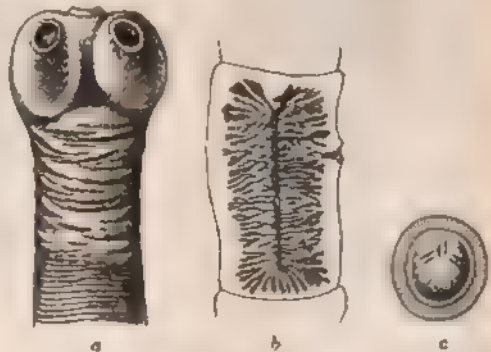


FIG. 115. *Tænia saginata*: a, head much enlarged; b, developed proglottis; c, ovum (Schmidt and Strasburger).

may be both mediate and intermediate host, the cysticerci becoming attached to a number of organs.

It frequently happens that the *Tænia saginata* and *Tænia solium* do not cause any symptoms of disease, except that the segments are discharged. In a few cases there are abdominal pains, irregular evacuation of the bowels, vomiting, lassitude, mental depression, loss of appetite, and headache. These symptoms disappear after the expulsion of the parasite.

The *Bothriocephalus latius* is apt to induce more serious symptoms, even grave anemia similar to pernicious anemia. We r

Treatment.—Tapeworms cannot be removed by simple purgatives; they require anthelmintics. The action of these remedies has not as yet been entirely elucidated in all its details. They probably act as specific poisons on the protoplasm of the parasites. The fact that all of them are difficult of absorption from the intestine facilitates this effect.



FIG. 120.—*Bothriocephalus latus*. a, head and neck, b, c, d, e, f, segments taken from different parts, g, shrunken segments after the laying of the eggs. (Guart.)

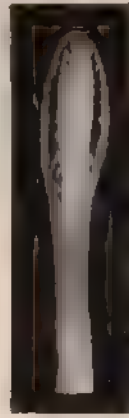


FIG. 121.—Enlarged head of *Bothriocephalus latus*. (Guart.)



FIG. 122.—Transverse section of head of *Bothriocephalus latus*. (Guart.)

The most extensively employed tapeworm remedy is the oleoresin of the root of the male fern, *oleoresina aspidii*. The active principle is the filicic acid, which is readily converted into an inactive crystalline anhydrid. This transformation takes place particularly in old roots, and explains why the extract from fresh roots is more active. The oleoresin of male fern is poisonous, and it is therefore necessary to insist on exact dosage; cases of fatal poisoning have been observed, preceded by convulsions, visual disturbances, blindness, paralysis, and diarrhea. The adult dose of *oleoresina aspidii* is 2 to 8 Gr. (5ss. ij.); of the root itself, about 20 Gm. (Sensitive patients may take the oleoresin in gelatin capsules.

Filmaron is the trade name for the isolated active principle of the root of male fern. The dose for adults is 1 Gm. (15 grains), for children 0.3 to 0.5 Gm. (5 to 8 grains).

FIG. 123



FIG. 124



FIG. 125



FIG. 126



FIG. 127



FIG. 128



Hymenolepis nana. FIG. 123 body. 124 natural size. 125 head. 126 hooklets. 127, eggs. 128 egg, magnified 600 times. (From Mosler.)

Cortex granati is used nearly as frequently as male fern. It is the bark of a pomegranate tree (*Punica granatum*). The active

constituent of the bark is the alkaloid pelletierin. Pelletierin itself has been employed as an anthelmintic, with satisfactory results. To diminish its solubility and thus prevent its absorption, tannic acid is sometimes added to it.

	Gm. or Gs.	
R—Pelletierini	0.8	gr. xij
Acidi tannici	0.5	gr. viiss
Aque	30.0	℥j
Misce		

Sig = To be taken in one dose in the morning, and a tablespoonful of castor oil two hours later.

Pumpkin seeds (*Cucurbita pepo*) are particularly adapted for the treatment of children. Sixty to ninety grams (2 to 3 ounces) of the seeds, deprived of integument, are triturated thoroughly with sugar, and given mixed with milk. The dose is to be taken the first thing in the morning on an empty stomach. Two hours later a teaspoonful to a tablespoonful of castor oil is given.

All the above medicaments are effective only when fresh. The active ingredients undergo decomposition after a time. Anthelmintics are nerve poisons, and it is therefore necessary to be particularly careful and exact in the dosage.

Systematic treatment should be undertaken only after the physician has ascertained positively that a tapeworm is actually present by the demonstration of its ova in the feces or by the finding of isolated segments.

The attempt to expel a tapeworm from the intestine is frequently unsuccessful. The commonest cause of failure is that the intestine has not been thoroughly evacuated beforehand. To secure the best action of the anthelmintic, the bowel should be as nearly empty as possible, not only of food but also of the large amount of mucus which is usually present.

Before an anthelmintic is administered, at least two days should be devoted to the preparation of the patient for the treatment. The patient should give up his business and attend strictly to the preparatory treatment. The diet should be liquid: milk, not more than a quart; beef tea, and coffee if desired. During these two days the patient should receive a dose of magnesium sulphate 4 Gm. (℥j) three times a day, in water, so that the upper part of the intestine may be thoroughly cleansed, especially of mucus. This may also be accomplished by a single dose of calomel, 0.3 to 0.5 Gm. (5 to 8 grains), or two tablespoonfuls of castor oil at night. The next morning the patient should take the oleoresin aspidium; as a rule, a dose of 8 Gm. (℥ij) is large enough for an adult. This dose should be taken in its entirety, best in cold black coffee, followed in one or two hours by a vigorous purge, preferably castor oil in the dose of two tablespoonfuls.

The patient may take half the prescribed dose of oleoresin, an hour later a tablespoonful of castor oil, in another hour the balai

of the oleoresin, and again, an hour later, a second tablespoonful of castor oil. After the administration of male fern and castor oil a few cases of poisoning have been observed, and it has been assumed that the castor oil had assisted the poisoning. The filicic acid in the male fern is said to be soluble in oil and therefore more likely to become absorbed. This effect of castor oil has not, however, been conclusively demonstrated. It is more probable that in these cases the filix alone was to blame, without the castor oil, since impure male fern is particularly apt to cause poisoning. Nothing can be adduced against the administration of castor oil after male fern. There is, of course, no objection to substituting calomel or bitter mineral water for the castor oil. In order to reduce the quantity of oleoresin necessary for a cure, the medication has been administered combined with chloral hydrate, 2 Gm. (3ss) of the former to 1.5 Gm. (22½ grains) of the latter, followed by a powerful drastic purgative, and good effects have resulted.

Very sensitive persons may take the oleoresin aspidium and the castor oil in gelatin capsules, in order to prevent loss of the remedy from vomiting.

Oleoresin of male fern is given to children, according to age, in doses of 1 to 4 Gm. (15 to 60 grains).

ASCARIS LUMBRICOIDES (ROUND WORM).

A parasite that frequently infests the human intestine is the *Ascaris lumbricoides* (Fig. 129), or round worm, belonging to the family of Nematodes. The round worm is of a reddish or brownish color, about $\frac{1}{4}$ inch in diameter—the size of a small goose-quill. The male varies in length from 4 to 8 inches, the female from 6 to 12. The male is the more curved, the female the straighter of the two. It has been estimated that the genital tubes of a large mature female ascaris may contain 60,000,000 eggs. The eggs after passing from the intestine are exceedingly tenacious of life; they may survive as long as two or three years. The ova may be taken into the stomach, where in the course of one, two or three weeks the worm is hatched out. The eggs are characteristic; their interior consists of a granular mass, surrounded by a thick double shell and an albuminoid coating (Fig. 129, C).

Infection usually takes place by way of the patient's mouth. An intermediary host is not necessary.

Symptoms.—In many cases no symptoms whatever are produced by the *ascarides lumbricoides*. Occasionally abdominal pains and nervous symptoms are present as in cases of tapeworm.

Diagnosis.—As an aid to diagnosis it should be remembered that an increased number of eosinophiles in the blood indicates possible infection with animal parasites. When this is found in the course of routine examination, we should institute a search for the eggs or

embryos of parasites in the feces. De la Fuente points out two signs which establish the diagnosis of intestinal helminthiasis without examination of the stools. One is the occurrence of colicky attacks coming on very suddenly, seizing the child in the midst of play, quite severe at the outset, and confined to one part of the abdomen; all the rest of the abdomen may be palpated without causing the slightest pain, but the moment the seat of the colic is touched the child will cry. The other is bilateral narrowing of the visual field, usually so pronounced as to be detected by passing the finger to and fro before each of the patient's eyes.

Couillaud has described a specific sign of the tongue found in helminthiasis. In cases of ascarides and oxyuris the fungiform papillae are hypertrophied. At the base of the tongue are seen scattered red points which are also diffused along the sides and at the tip.

In rare instances grave complications are induced by a round worm perforating the intestine. Should one become incarcerated in the ductus choleloechus or the gall bladder, grave jaundice and cholangitis might follow (see page 616).

The evidence shows that the ascaris is able to perforate the intestinal wall, especially when favored by tuberculous, typhoid or other ulcerative lesions. The biliary system also should be borne in mind, particularly in the case of children or adults who are known to have worms and who have at the same time chronic jaundice, convulsions, fever, violent pain in the region of the liver, or symptoms of hepatic abscess.

Treatment. The remedy most frequently employed and with the



FIG. 129. *Ascaris lumbricoides*. A, female; B, male; C, egg; a, female genital opening; b, the spermatheca; c, the enlarged cephalic extremity with its three spicules. (After Perlo from Ziegler.)

greatest expulsive efficacy is santonin. Santonin is a nerve poison and in large doses induces sensory disturbances, especially of the eye, violet and yellow colors, hallucinations, lowering of temperature, lassitude, convulsions, and paralysis of respiration. The urine, after santonin has been taken, is greenish-yellow and contains a substance which turns purple-red on the addition of concentrated sodium hydrate solution. This color does not fade on the addition of ether, thus differing from the urine pigmentation that follows the use of rhubarb and senna. An important differential point is thus afforded in the diagnosis of poisoning by santonin.

The dose of santonin for children between the ages of one and eight years is 0.01 to 0.03 Gm. ($\frac{1}{4}$ to $\frac{1}{2}$ grain), not exceeding 0.06 to 0.1 Gm. (1 to 2 grains) in one day. The maximum single dose for adults is 0.1 Gm. (2 grains); the maximum amount per day, 0.5 Gm. ($7\frac{1}{2}$ grains). Usually the first dose is given early in the morning, and in case round worms are expelled the medication is continued during that day and part of the next. Santonin is best given in powder with castor oil:

	Gm. or Ce.	
R—Santonini	0 1	gr. ij
Olei ricini	15 0	3ss
Misce.		
Sig.—One dessertspoonful (warmed) to be taken in the morning.		

Or it may be combined with calomel:

	Gm. or Ce.	
R—Santonini	0 3	gr. v
Hydrargyri chloridi mitis	0 06	gr. j
Sacchari albi	1 5	gr. xxij
Misce et ft. pulv. no. iii.		
Sig.—Three powders to be taken within three hours in the morning.		

There are also santonin tablets (trochisci santonini), each containing 0.03 Gm. ($\frac{1}{4}$ grain), which may be given to either adults or children. Give castor oil a few hours later.

On account of its non-toxic properties, oleum chenopodii (American wormseed) is warmly recommended in place of santonin. It is said to be equally efficacious and free from the disadvantages that pertain to santonin. The oil is administered either pure or in the form of an emulsion:

	Gm. or Ce.	
R—Olei chenopodii	5 0	5j
Misce.		

Sig.—Eight to fifteen drops to be taken in sweetened water two or three times, at intervals of one hour.

	Gm. or Ce.	
R—Olei chenopodii	5 0	5j
Pulvis traccanthæ	33 3 0	3j
Aquæ destillatæ	33 45 0	3iss
Syrum auranti		
Misce et ft. emulsiō		

Sig.—One dessertspoonful to be taken two or three times within two hours.

As the oil has a nauseating taste, it is advisable to follow the dose with a sip of milk, sweetened water, or dilute raspberry juice. One or two hours after the oil has been taken, a purgative should be administered.

OXYURIS VERMICULARIS.

(*Pin Worm*—*Thread Worm*—*Seat Worm*—*Maggot Worm*—*Wiltail*.)

Oxyuris vermicularis is the well-known small white thread worm. The males are 4 millimeters ($\frac{1}{4}$ inch) long, and the females 9 to 12 millimeters ($\frac{3}{8}$ to $\frac{1}{2}$ inch) (Figs. 130 and 131). The worms occur in patients of all ages, but most often in children. The infection is brought about by ingestion of the ova (Fig. 132), the patient's fingers harboring the parasite, to which the ova adhere, and thus contaminating the food, so that entire families are occasionally infected. The period of development of the worms in the stomach is about five weeks. The young worms here escape from the eggs and migrate into the small intestine, where they grow to sexual maturity. Reproduction occurs in the lower portions of the small intestine, in the cecum, and in the vermiform appendix. The largest quantity of thread worms is found usually in the cecum and appendix. The ova develop in the fertilized female, and the latter migrates downward through the large intestine as far as the rectum, where she deposits her eggs either upon the feces or upon the mucous membrane; the larger portion, however, is either deposited outside of the intestine or carried there—in the neighborhood of the anus and the perineum. After depositing her eggs the female dies. It seems as if these worms were capable of penetrating into the mucous membrane of the small intestine, especially when this membrane is pathologically altered, dying there and undergoing calcification. According to rather recent researches, calcareous nodules about the size of a pin's head are found in the intestinal mucous membrane, corresponding to the follicles of Peyer's patches and the solitary follicles, at the bottom of which the oxyurides are found. According to modern views, these parasites may cause appendicitis (see page 767).

Symptoms.—When the worms are numerous the symptoms are apt to be quite pronounced. There is a most tormenting itching in the rectum and in the region of the anus, which occasionally renders the patients quite desperate. This itching is probably induced by the movements of the migrating worms, and is particularly violent at night from the warmth of the bed. As the pruritus and becomes intolerable the patients endeavor to relieve themselves by scratching the itching spots. Both children and adults indulge in this habit with equal energy, and as a result excoriations of the skin are frequently found in the anal region. In girls the oxyurides may creep into the vagina, inducing vaginitis, with itching, leading occasionally to masturbation.

There may be other symptoms, as pains in the abdomen, capricious and sometimes ravenous appetite (in spite of which the child becomes thin and sallow), grinding of the teeth at night, picking of the nose, nausea, dizziness and other reflex phenomena, irregularity of the bowels, or diarrhea.

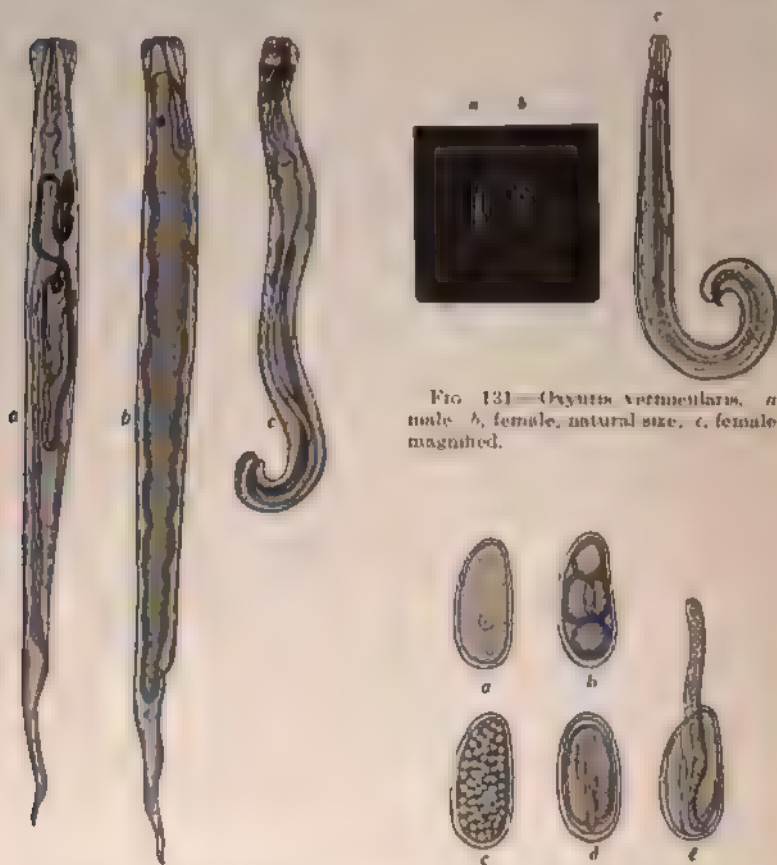


FIG. 131.—*Oxyuris vermicularis*. *a*, male; *b*, female, natural size; *c*, female, magnified.

FIG. 130. *Oxyuris vermicularis*. *a*, sexually mature female; *b*, female filled with eggs; *c*, male $\times 10$ (After Heller, from Ziegler.)



FIG. 132. Eggs of *Oxyuris vermicularis* in various stages of development. *a*, *b*, *c*, division of the yolk; *d*, tadpole-like embryo; *e*, worm-shaped embryo. $\times 250$. After Zenker and Heller, from Ziegler.

Diagnosis. The diagnosis is established by the discovery of ova of the *Oxyuris vermicularis* in the feces or by demonstration of the expelled worms themselves. It is frequently necessary to examine the material collected under the finger-nails of children, as it may be possible thus to demonstrate microscopically the eggs or parts of the worms.

Treatment. The object of the treatment is to remove the young brood from the small intestine, to clear the large intestine of adult parasites, and to protect the patient from fresh infection. The removal of the young worms is accomplished by purgatives or vermifuges, and the cleansing of the large intestine is attained by appropriate enemata. The more rapidly the treatment is carried to completion, the better it is for the patient; short treatments generally give better results than protracted ones. The following five days' treatment for adults, as outlined by Zinn, gives excellent results:

First Day: A light fluid or semisolid diet. In the afternoon at three o'clock the following is to be taken:

	Gm. or Cc.
R - Hydrargyri chloridi mitis,	
Pulveris jalapæ	ss 0/5 gr. viij
Misce.	
Sig.—To be taken at three o'clock.	

At 6 o'clock an enema of 1 to 2½ liters (quarts) of warm 0.2-per-cent. to 0.5-per-cent. solution of soap (U. S. P.). By this method the entire gut is thoroughly cleared and well prepared for the action of the vermifuge.

Second Day: Liquid diet, with some buttered rolls. Early in the morning one cup of black coffee. In the forenoon at 8, 10 and 12 o'clock, a powder containing 0.05 Gm. (1 grain) santonin and 0.1 Gm. (2 grains) calomel. At 2 o'clock in the afternoon two tablespoonfuls of castor oil, to be repeated if necessary at 4 o'clock.

Third Day: Liquid and semisolid diet. Early in the morning a warm full bath. In the forenoon and in the afternoon an enema (in the left lateral or in the genupectoral position) of 1 to 2½ liters (quarts) of the soap solution mentioned above.

Fourth and Fifth Days: Exactly as on the third day. In the evening a warm bath.

Before and after every meal the hands should be carefully washed with warm water, soap and brush, and disinfected with alcohol or a 1:3000 sublimate solution. The same procedure should be followed out after each defecation, and the region of the anus should always be carefully washed. The medicines are to be taken between the meals. In order to prevent continued reinfection, the patient is to be carefully instructed about the mode of the infection. During and after the treatment the underwear and the bedclothes should be frequently changed.

Apart from santonin, any of the other vermifuge medicaments may be employed as described under Treatment of *Ascaris Lumbricoides*. The doses of the anthelmintics and the size of the enemata must be modified according to the ages of young patients. The pruritus of the anus and its neighborhood may be treated by inunctions of mercurial ointment. (See Chapter LIV on Pruritus Ani.)

The soap enemata may be replaced by weak infusions of quassia, lime-water, 9-per-cent. acetic acid, 20-per-cent. vinegar-water, cod-liver oil, naphthalin, 0.5-per-cent. tannic acid solution, salt solution, glycerin-water, or thymol oil (thymol 1 to olive oil 100). It is also advisable to add anthelmintics to the enemata. For this purpose oleoresina aspidii may be especially recommended; it is triturated with warm thin oatmeal gruel, and sufficient gruel is gradually added to make 1 to 1½ liters (quarts) for an enema.

The diet during the treatment should be semiliquid, as mentioned above. Raw carrots are popularly regarded as an adjuvant in the expulsion of worms.

The treatment as here detailed is usually effectual in five days.

In young children the administration of the enemata is occasionally troublesome, and on this account the treatment may have to be continued a few days longer. If ova and single specimens of the parasite are again found, the treatment may be repeated once or twice after a week or two.

ANKYLOSTOMA DUODENALE; UNCINARIA AMERICANA.

(*Uncinariasis*—Hookworm Disease—*Necator Americanus*—*Miner's Anemia*—*Strongylus Duodenalis*—*Dochmius Duodenalis*—*Uncinaria Duodenalis*—Ground-itch Anemia.)

The *Ankylostoma duodenale* is a native of Egypt and Japan, which later made its appearance in Italy. It has also been found in the last few years in Germany, particularly in the mining districts. The male is yellow, 7 to 10 millimeters ($\frac{1}{4}$ to $\frac{1}{2}$ inch) long; the female brown, 10 to 18 millimeters long (Figs. 133 and 134). The prehensile apparatus is at the cephalic extremity, and the worms attach themselves by hooking upon the mucous membrane of the intestine. The infection is usually brought about by transference of the eggs through contaminated hands; or the larvæ enter through the skin (Fig. 135), causing a condition known in the South as "ground itch," "foot itch," "toe itch" and "dew itch." In the experience of Stiles, 87 per cent. of hookworm cases definitely admit a history of ground itch. The worm thrives in the jejunum and the upper portions of the ileum, and rarely in the duodenum. It feeds on the blood of the host. According to more recent observations the worm is said to actually eat the intestinal epithelium.

At the fifth annual meeting of the American Gastroenterological Association in 1902, Charles Wardell Stiles,¹ of the Public Health and Marine Hospital Service, called attention to a new species of parasitic hookworm, which he named *Uncinaria americana*. It differs from *Ankylostoma duodenale* (Fig. 136) chiefly in the following characteristics: Ventral recurved hook-like teeth are

¹ Stiles, A New Species of Hookworm (*Uncinaria americana*) Parasitic in Man, *American Medicine*, May 10, 1902, p. 777

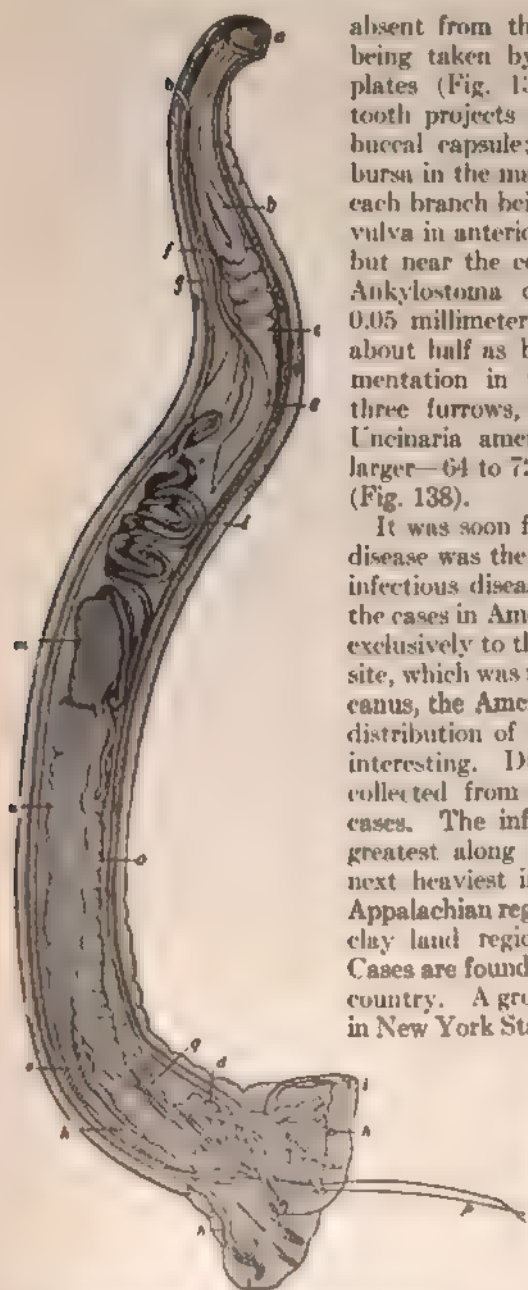


FIG. 133. Male of *Ankylostoma duodenale*. a, head; b, esophagus; c, gut; d, anal glands; e, cervical glands; f, skin; g, muscular layer; h, excretory pore; i, tri-lobed bursa; k, ribs of bursa; l, seminal duct; m, semina; n, ductus ejaculatorius; o, its groove; p, penis; q, penile sheath. Magnification 20. (After Schulthess, from Ziegler.)

absent from the mouth, their place being taken by a pair of semilunar plates (Fig. 137); a dorsal conical tooth projects prominently into the buccal capsule; dorsal ray or caudal bursa in the male divided to its base, each branch being bipartite at its tip; vulva in anterior half of female body, but near the equator. The eggs of *Ankylostoma duodenale* are about 0.05 millimeter ($\frac{1}{200}$ inch) long and about half as broad, and show segmentation in the shape of two or three furrows, whereas the eggs of *Uncinaria americana* are somewhat larger—64 to 72 by 36 to 40 microns (Fig. 138).

It was soon found that hookworm disease was the most common of the infectious diseases in the South, and the cases in America were due almost exclusively to this newly found parasite, which was named *Necator americanus*, the American murderer. The distribution of the parasite is rather interesting. During one week Stiles collected from correspondence 6858 cases. The infection is heaviest or greatest along the sand area. The next heaviest infection occurs in the Appalachian region, and in general the clay land regions are less infected. Cases are found in other parts of the country. A group of cases was found in New York State, all the individuals



FIG. 134. *Ankylostoma duodenale*, male and female. Natural size. (From Mosler.)

being United States soldiers who enlisted in the Southern States. Two patients were from Connecticut.

Stiles visited about one hundred and thirty cotton mills and eight milling camps in the South, and found many men, women and children in the mills who were infected with hookworm disease to such an extent that the diagnosis could be made without the aid of the microscope. One out of every eight of the cotton mill employees of the Southern States, if we accept these one hundred and thirty as fairly representative, has hookworm disease so obviously that the microscope is not needed in the diagnosis. The distribution of the disease bears an inverse relation to the distribution of what one might call the efficiency of the cotton mill labor. If one enters a

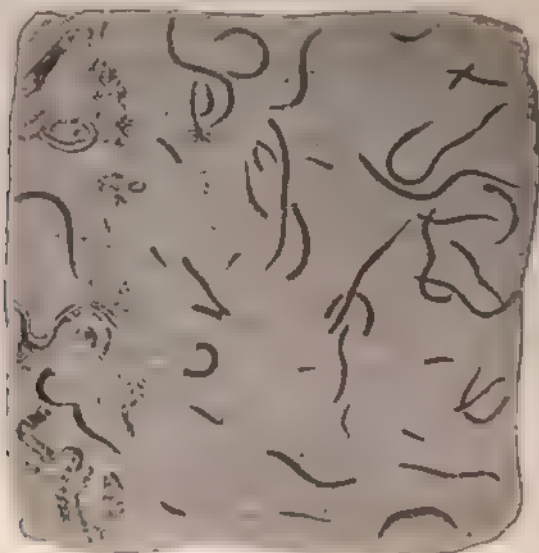


FIG. 125. Section through the skin of a dog within two hours after it had been infected with the hookworm, *Ankylostoma duodenale*. (Greatly enlarged. Stiles.)

Piedmont mill he will see that the laborer is distinctly more efficient than the laborer in a mountain mill or a sand-land mill. In the sand-land mills the infection among the employees runs up to as high as 50, 60 or nearly 70 per cent. in the men, women and children employed, while in some of the Atlanta mills it drops to 5 per cent. or even to zero. Atlanta is in a clay belt, and the inhabitants live under good sanitary conditions.

Hookworm disease, as found in the United States, has been traced to the west coast of Africa, even as far as the pigmy tribes. Unquestionably the negroes must have brought many hookworms to this country. The disease is an African one, which has been transmitted to the whites. The negroes, having had the disease for generations, do not suffer from it so intensely as do white people.

They are ambulant cases, carriers of the infection, and of necessity there is more hookworm disease in localities where negro population predominates largely. Seventy-nine per cent. of the negro farm houses which Stiles examined and tabulated in North Carolina, South Carolina, Georgia, Alabama, and Mississippi, had no privy connected with them. These carriers of the hookworm suffer comparatively little themselves, but serve as breeders of the worm and sowers of its seeds, to the lasting injury of their white neighbors. Cuba and Porto Rico have many cases of *uncinariasis*, and thousands of cases are reported from the Philippines.

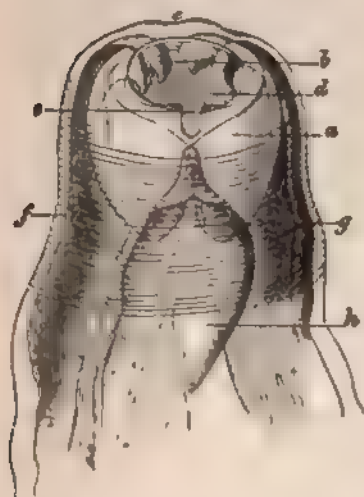


FIG. 136. Head of *Ankylostoma duodenale*. a, buccal capsule; b, teeth of capsule; c, teeth of dorsal margin; d, oral cavity; e, ventral prominence; f, muscle layer; g, dorsal groove; h, esophagus. (After Schulthess, from Ziegler.)



FIG. 137. Head of *Uncinaria americana* showing the buccal cavity and semilunar plates. (Guarni.)



FIG. 138. Eggs of *Ankylostoma duodenale*. a, d, various stages of segmentation; e, f, eggs containing embryos. Magnification 200. (After Ferronetti and Schulthess, from Ziegler.)

The female hookworm lays an immense number of eggs. These eggs are excreted with the feces of the patient. As many as 4000 worms have been found in one patient, and as many as 2,000,000 ova may be discharged in a single stool. Where proper conditions of soil, climate, moisture and oxygen prevail, these eggs readily hatch into larvae. The larvae are exceedingly minute, unseen by the naked eye; they infect the soil, drinking-water, and food. It has been demonstrated that the eggs and larvae may be carried on the legs of flies.

Bentley proved hookworm infection to be due to the entrance

of the larvæ of the ankylostoma into the skin. Loos¹ also proved that the larvæ could penetrate human skin, producing a local reaction, and that men and dogs could acquire the disease by the cutaneous route. Boycott² produced an infection by applying a few full-grown larvæ to the skin of the forearm for a couple of hours. There was a slight local reaction and some itching, and eggs were found in the stools after fifty days. The larvæ used for the experiment were a single batch bred for the purpose from infected feces. Of the possibility of larvæ passing through the skin, and ultimately reaching the bowel, there is therefore no doubt (Fig. 135). The infection of the feet is from contact with the soil, which becomes contaminated by the deposition of the laborer's evacuations. The larvæ present in the skin produce an itching sensation accompanied by an inflammatory reaction, and sometimes secondary infection with other microorganisms may take place. This condition is commonly known as ground-itch or dew-itch. The larvæ, having pierced the skin, soon gain entrance to the lymph and blood channels, through which they are carried to the heart and thence to the capillaries of the lungs. They then pass through the capillary walls into the alveolar spaces; from here they migrate through the bronchioles into the bronchi, and then crawl up the trachea to reach the mouth cavity, after which they are swallowed in ordinary acts of deglutition, eating, drinking, etc., and, according to Lindeman,³ thereby reach the intestinal tract.

Pathology.—In hookworm disease the jejunum and particularly the ileum are the seat of a severe catarrhal process, which also affects to a degree the other portions of the intestine. There is a large amount of mucus in the intestinal canal, in the walls of which the worms are embedded, and which is often blood-stained in places. The lesions of the intestine are confined to the mucosa, and there is often degeneration and atrophy of the intestinal and gastric mucous membrane. At the point of attachment of the worms there is a tiny erosion, superficial, not deep, and about one-half millimeter in diameter. This erosion is usually not surrounded by any discoloration, and is difficult to locate with the naked eye.

Polycythemia and polychromatophilia are present typically in the severe cases. One of the most remarkable features of hookworm disease is the utter lack of agreement between the severity of symptoms and lesions, including the blood changes, and the number of worms found in the intestine Evans⁴. In some of the more severe cases a decided leukopenia rather than leukocytosis is present. In the differential count of white cells, apparently

¹ *Annals of the American Academy of Medicine*, 1906, June, 704.

² *Annals of the American Academy of Medicine*, 1906, June, 704.

³ *Annals of the American Academy of Medicine*, 1906, June, 704.

⁴ 1903.

⁵ *Annals of the American Academy of Medicine*, 1906, June, 704.

the only important abnormality is in the number of eosinophiles. Eosinophilia is an almost constant finding, and practically all observers are agreed that the degree of eosinophilia is not an index of the severity or extent of the infection.

Symptoms.—The principal effect produced by these parasites is a grave disease that runs its course under the picture of pernicious anemia, accompanied by weakness of the heart, dizziness, dyspnea, and an occasional slight fever. Unless the worms are removed, the poverty of the blood may reach such a stage as to lead to a fatal outcome. It is as yet undetermined whether the anemia is caused by loss of blood or by hemolytic substances derived from the parasites.

Besides the anemia and its concurrent symptoms, there are gastric disturbances, pyrosis, abdominal pains, nausea and sometimes vomiting. In the feces occult blood is always found, and often the Charcot-Leyden crystals. Not every host of this parasite is necessarily sick; examinations of a large number indicate that the great majority do not exhibit any symptoms of the disease.

On the other hand, Stiles¹ found that if the patient is infected before puberty his physical and mental development is retarded and he shows a more or less extreme anemia. A person twenty-one years of age may appear not better developed than one fourteen to eighteen years old. The skin is dry, and there is a noticeable absence of perspiration. The color may be waxy white to dirty yellow; it has a resemblance to tallow. The hair of the head is dry, reminding one of hemp. The beard and the axillary and pubic hair may be very late and scant of growth. Edema may be present in the face, feet, legs, scrotum, or entire body; it seems to be especially common over the cheek bones. Skin wounds are likely to be rather slow in healing. Many of the patients (about 57 per cent. of the well marked ones) either show tibial ulcers or give a history of such lesions. The face is likely to have an anxious, oftentimes stupid expression. Dark lines under the eyes are common. The visible mucous membranes may be chalky white. The pupils show a tendency to dilatation, even when facing a strong light; many patients exhibit a peculiar blank stare; night-blindness is reported in a number of instances. Cervical pulsation may be very prominent, and is frequently visible six to twelve feet distant. The thorax may be so emaciated that the ribs stand out very prominently. The shoulders droop and are thrown forward; the shoulder blades stand out prominently (winged shoulder blades). In many instances the abdomen is so protuberant as to remind one of pregnancy. This condition is known locally as "pot-belly," "buttermilk-belly," or "shad-belly."

Diagnosis. There is probably no disease known to the medical profession which is more easily diagnosed and more easily treated

¹ Hookworm Disease, Public Health Bulletin, No. 32.

than hookworm disease. In the diagnosis the color of the stools may be suggestive, as hookworm patients pass reddish-brown stools. The following simple test may be made: Several ounces of the stool are wrapped in white paper and allowed to stand for a few hours. In severe and in some moderately light cases a distinct reddish-brown stain will be found on the paper. It is rare that the adult worms are seen in the discharges except during treatment, but the stools of hookworm cases contain the characteristic eggs of the parasite, and by finding these eggs under the microscope a positive diagnosis can easily be made.

Treatment.—Ground itch in the papular or vesicular stage may be treated with 5-per-cent. salicylic acid suspended in collodion. This usually limits the attack to one or two days. During the pustular stage the wound should be cleansed and cauterized with silver nitrate, and then a dry dressing of zinc oxide ointment applied twice daily. To allay itching and prevent secondary infection a combination 5-per-cent. zinc oxide and salicylic acid ointment applied locally, twice daily, is recommended. It is important to keep the affected foot bandaged or covered to prevent scratching or rubbing.

To expel the worms after a preparatory treatment with purgatives, thymol gives the best results. It is more effective when mixed with an equal quantity of sodium bicarbonate; its value is enhanced and the unpleasant stomach symptoms relieved. Magnesium sulphate is first given, to remove the mucus and feces surrounding the hookworms. The night before the initiatory treatment, 15 Gm. ($\frac{1}{2}$ ounce) of magnesium sulphate is to be taken in two or three doses at intervals of one hour; the next morning two doses of thymol, in capsules, two hours apart; two hours later, 15 Gm. ($\frac{1}{2}$ ounce) of magnesium sulphate. Never give castor oil. Oils of all kinds, fats and alcohol favor the absorption of the thymol, with toxic effect. The dose of thymol should be adapted to the age of the patient, as follows: Under five years, 0.5 Gm. ($7\frac{1}{2}$ grains); five to ten years, 1 Gm. (15 grains); ten to fifteen years, 2 Gm. (30 grains); fifteen to twenty years, 3 Gm. (45 grains); twenty to sixty years, 4 Gm. (60 grains); over sixty years, 3 Gm. (45 grains). The patient should lie on his right side, which assists the thymol in entering the duodenum. No food or liquid is allowed until after the last dose of magnesium sulphate has worked off thoroughly. This treatment should be repeated once a week until microscopic examination shows that the ova are absent from the feces. Another way to check up the results of the treatment is to instruct the patient to wash his stools for three days through a cheesecloth, keeping the cloth constantly moist. The fecal matter washes through, and the worms are found in the cheesecloth. The treatment is to be continued as long as the worms are found.

Should thymol fail to effect a complete cure, or should the patient seriously object to it on account of the burning sensation that attends its administration, oil of chenopodium, American wormseed

may be prescribed. It is highly recommended by Oriental physicians who have had extended experience with it. After the patient has fasted for eight hours, 30 Gm. (1 ounce) of magnesium sulphate is to be given. Two hours later 1 Cc. (15 minims) of oil of chenopodium is administered on sugar, and repeated every two hours for three doses. The last dose is to be combined with 30 Cc. (1 ounce) of castor oil and 3 Cc. (45 minims) of chloroform. The treatment is to be repeated at intervals of one week until no more ova are found in the feces. Smaller doses are advocated for children.

The duodenal tube is used for introducing the vermifuge directly into the intestine (see Chapter III). Instead of 34 per cent. of cures, as in the case of a first mouth treatment, Kantor¹ reports that fully 80 per cent. are cured by the first tube treatment. Only one repetition is necessary for full relief in the great majority of cases. Three cubic centimeters (45 minims) of the oil of chenopodium are injected with a glass syringe. Ten minutes later, 100 Cc. (3 ounces) of a saturated solution of magnesium sulphate is introduced through the tube into the intestine by gravity. The flush removes the drug quickly from the small intestine and prevents undue toxic effects. The patients have a copious watery movement of the bowel in a half-hour which contains the oil and worms.

Oil of eucalyptus is preferred by some authors. It can be combined with castor oil and chloroform.

	Gm. or Co.	
R—Olei eucalypti	3 0	℥xliv
Chloroformi	2 0	℥xxx
Olei ricini	30 0	℥j
Misc.		

Sig. —To be taken on arising in the morning, every other day for ten days.

One course of treatment frequently proves effectual. Should ova be found in the feces, another ten days' treatment will be required.

The oleoresin of male fern is employed either in a single portion of 8 Gm. (3 ij) or in doses of 4 Gm. (5 j) each on successive days. Castor oil or calomel is given three hours after the administration of the anthelmintic. Filmaron may also be employed.

After the expulsion of the worms it is well to administer iron to hasten the recovery from the anemia. Pilule ferri carbonas (Blaud) 0.3 Gm. (5 grains) may be administered three times a day. The tincture of iron chlorid may be given diluted in water in doses of 0.3 to 0.6 Cc. (5 to 10 minims) after meals. The hypodermic administration of the sterilized solution of citrate of iron, supplied in glass ampoules, is often found a valuable aid to prompt recovery (see page 581).

Worm-carriers who have no symptoms of illness should be treated prophylactically. During epidemics and as a prophylactic measure the laborers in mines should be instructed as to how the infection is brought about, compelled to practice scrupulous cleanliness, and taught to disinfect their feces.

¹ John L. Kantor: The Cure of Hookworm Infection, American Journal of the Medical Sciences, April, 1920

TRICHOCEPHALUS DISPAR.

This worm, also called whip worm (Fig. 139), inhabits the cecum and colon of man and may penetrate into the mucous membrane,



FIG. 139.—*Trichocephalus dispar*. a, male, b, female. (From Moslet.)

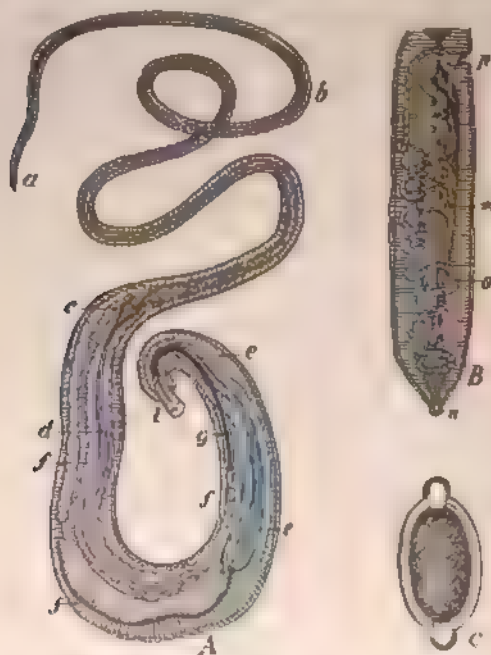


FIG. 140. *Trichocephalus dispar*. A, male B, posterior extremity of female; f', ovum. a, head b, cephalic extremity of body with esophagus c, stomach d, gut. e, clitellum f, seminal canal, g, penis h, bell-shaped penile sheath, with tip of penis i, m, gut of female, n, anus, o, uterus p, vaginal cleft. Magnification, 10. (After Küchenmeister and Zörn, from Ziegler.)

abstracting blood therefrom. The ova are lemon-shaped (Fig. 140, C). As a rule the worm causes few subjective symptoms; occasionally, however, it gives rise to severe enteritis, anemia, fever, or meningeal manifestations. The prevalence of whip-worm

infection in a locality is, to a certain extent at least, an index of the intelligence and cleanliness of its citizens. The parasites are difficult to destroy, on account of the fact that they thrust their attenuated head ends through folds of the mucosa, and so have a very good hold on the bowel. In addition to the discovery of these worms in their usual habitat, they have been found by various authors in the ileum, the vermiform appendix, and the peritoneal cavity.

Treatment.—Tapeworm remedies have been used successfully. It may become necessary to use enemata containing petroleum—4 to 15 Cc. (3j-iv) to a liter (quart) of water, frequently repeated.



FIG. 141.—Female of *Anguillula intestinalis*, with eggs and embryo. (After Perroncito, from Ziegler.)

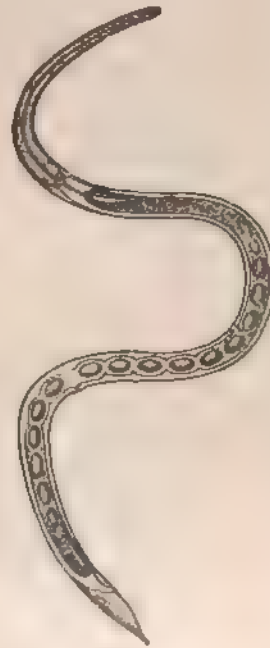


FIG. 142.—*Anguillula intestinalis*. (After Braun, from Ziegler.)

ANGUILLULA INTESTINALIS (*STRONGYLOIDES STERCORALIS*).

Anguillulae (worms 1 to 2 millimeters in length—Figs. 141 and 142) are found in the tropics and semitropical countries, frequent in association with *Ankylostoma duodenale*. It seems to

been conclusively shown that "*Anguillula intestinalis*" and "*Strongyloides stercoralis*" are synonyms for the same parasite. The cephalic extremity is rounded, and pierced by the mouth, about which there are three or four papillæ. Next to the mouth there is a vestibule; then comes the esophagus, which contains three horny teeth. Beyond the esophagus the intestine extends to the anus, which is situated at the base of the tail. The female is larger than the male and usually more numerous. The vulva is situated on the right side of the body, a little below the middle, and leads into a double uterus, each horn of which ends in an ovary. The male parasite presents two cone-shaped and curved spicules at the base of the tail, which serve as the copulatory organ. The eggs are small, elliptical, and are usually segmented when laid. There are some anatomic differences between the free-living generation, the parasitic form, and the larvæ.

Symptoms.—The worms may be present without symptoms, or they may give rise to serious conditions, in particular to diarrhea, which is known under the name of anguilluliasis.

Treatment.—The anthelmintics mentioned under "*Trichocephalus*" are also employed here. Recently glycerin has been recommended: 25 Gm. (3vj) of pure neutral glycerin by mouth, and immediately afterward another 25 Gm. in keratin capsules to retard absorption; again, two hours later, 30 Gm. (3j) per rectum. This treatment is to be given twice weekly. Olive oil in large doses is also said to be effectual.

TREMATODES.

Trematodes (flake worms), found quite often in the lower animals, are comparatively rare in man. They are leaf- or tongue-shaped, with an apparatus for sucking. A few isolated cases have been reported in the United States, but the subjects were foreigners. Trematodes are mostly confined to Japan, China, Egypt, Arabia, and India. The worms are hermaphrodite. There are several varieties: *Distoma hepaticum* (Fig. 143), *Distoma lanceolatum* (Fig. 144), *Distoma felinum*, *Distoma crassum*, *Distoma hematobium* (Bilharzia, Fig. 146), *Distoma heterophyes*, *Distoma conjunctum*, *Distoma sinense*, and some others.

Stiles has found the *Distoma hematobium* (Bilharzia) but twice in the United States, both times in foreigners. On the Isthmus of Panama, according to statistical records obtained from the Chief Sanitary Office, *Distoma hematobium* has been reported 104 times. These statistics were obtained from approximately 30,000 stool examinations. The worms differ from most trematodes by being two-sexed instead of hermaphrodite. They have a white appearance, are pointed at each end, and 1 centimeter ($\frac{1}{3}$ inch) long. They are all provided with suckers. When young the parasites live

apart; but after maturing the female enters the gynecophoric groove or canal of the male (Fig. 143), and frequently remains completely hidden there, though in some cases it protrudes, on account of its greater length, at the posterior extremity.

From observations made by Brayton¹ it appears that the portal vein is the principal habitat of the adult parasite, but the young



FIG. 143. *Distomum hepaticum* with male and female genital apparatus. From Ziegler, after Lauckert.



FIG. 144. *Distomum lanceolatum*. (v. Jaksch.)



FIG. 145. *Distomum hematobium* (Bilharz). (Günther.) A, male; B, female.

worms are found also in the liver, in the intestinal veins, in the bladder walls, and in the pelvic bloodvessels of both sexes. In the lungs the blocking of bloodvessels with the ova and surrounding

¹ Bilharziasis in the New World. Journal of the American Medical Association, April 30, 1910, p. 1437.

infiltrations gives rise to deposits, which may be mistaken for tuberculous deposits. Cirrhosis of the liver and biliary calculi may be caused by the presence of the parasite. Cystitis and urethritis are common complications, and often the parasite is the starting-point of stone. In the severer cases there may exist, with extension, the entire symptom-complex of "surgical kidney" or "surgical bladder." When the intestinal tract is involved, the symptoms resemble those of tropical dysentery.

Treatment.—From the above it would seem that the anthelmintics possess little value in the treatment of this disease. The treatment should be of a general character, to mitigate the symptoms, with special attention to the complications, for in many cases the disease is self-limited. Arsphenamine has been found to be absolutely useless and should not be employed.

Emetin intravenously, at intervals of two or three days, commencing with 0.02 Gm. ($\frac{1}{2}$ grain) and increasing rapidly to 0.06 Gm. (1 grain), has produced striking results. Ten to fifteen injections usually answer the purpose. This treatment is applicable to cases without infectious complications (see page 723).

The intravenous injection of antimony and sodium tartrate is now assumed to be a specific for bilharziasis. This drug also destroys the worms in the portal circulation. Both the subjective and the objective symptoms are immediately improved. Since the remedy is a powerful toxic drug, it must be used with great caution. During the first two weeks of treatment 0.03 Gm. ($\frac{1}{2}$ grain) is given intravenously every other day, and the dose is gradually increased until 0.12 Gm. (2 grains) is given at one injection. This amount can then be continued every three to five days until a maximum of 2 Gm. (30 grains) has been taken for the course.

Colloidal antimony sulphide intravenously is less toxic, and is said by some authors to be equally efficient in ridding the body of the infecting parasites. Rogers employed both the sodium-antimony tartrate and colloidal antimony sulphide, but found the latter to be entirely without evident benefit.

TRICHINA SPIRALIS.

Trichina spiralis belongs to the nematodes. The females are about twice the size of the males, which are 3 to 4 millimeters (about $\frac{1}{4}$ inch) long (Fig. 146). The parasite is usually taken into the stomach with uncooked pork, sausage, ham, or bacon. The envelope holding the embryo opens within three or four hours, liberating its contents. Fructification of the young parasites takes place in thirty to forty hours. The development of the trichinae in the intestine and the production of a new generation take place within one or two weeks. The young parasites are able to migrate into the muscles of the host and further develop (Fig. 147). The

appearance of the symptoms corresponds to the time consumed in the development of the trichinae and their penetration into the muscles. This disease is called trichinosis.

The symptoms are: loss of appetite, malaise, headache, fever, nausea or vomiting, diarrhea, tenderness of the epigastrium, cramps in the abdomen and limbs, great prostration, swelling and soreness of the muscles and indisposition to move them, increased rapidity of respiration, swelling of the eyelids and feet and sometimes of the knees, elbows, and ankles.



FIG. 146. *Trichina spiralis*. X female Y male a snout b mouth c body cells e embryo escaped from vulva i intestine n heart p papillae t testes u uterus v ventral vessels. Magnification, 80. E embryo greatly enlarged. (Gunn)



FIG. 147. Larvæ migrating trichinae in the muscles of man (enlarged). (Rosenheim)

The number of eosinophilic leukocytes in normal blood is 2 to 4 per cent. of the total leukocytic count, whereas in trichinosis the proportion may reach 40 to 50 per cent. after the fourteenth day. Eosinophilia must be taken in connection with other indications, since it is present in a variety of intestinal parasitic affections, in eczema and other cutaneous affections, in bronchial asthmatic conditions, and in typhoid fever.

The trichinae can rarely be found in the feces or blood of the patient. The best test is made by examination of the muscle. There is an instrument made especially for the purpose of removing

fragments of muscle tissue for examination. The trichinæ in the muscles become encapsulated and may undergo calcareous degeneration, followed by fatty degeneration, when recovery takes place. The disease is often fatal.

Treatment.—In the treatment it is advisable to thoroughly evacuate the bowels to remove the parasites which are still there. A dose of calomel, 0.3 Gm. (5 grains), should be given, followed in four hours by 15 Gm. (3ss) of magnesium sulphate. This can be repeated daily for four or five days to eliminate all the embryos. Thymol, 0.1 Gm. (2 grains), can be given as an anthelmintic and antiseptic four times during the twenty-four hours. Male fern or santonin may be administered if preferred. Glycerin has been prescribed to kill the worms by its hygroscopic properties; it may be taken in tablespoonful doses three times daily, well diluted. Large doses of quinin have been recommended. Picric acid has also been used.

A study of trichinosis in which beneficial results were attained in man by the use of serum from patients recovered from the disease has been published. In two patients in the active stages of trichinosis the administration of the serum showed remarkable curative power. Serum from experimental animals, however, convalescent from trichinosis, when injected into other animals or fed to them with trichinous meat, does not inhibit the customary development of trichinæ, but such serum is of decided value in combating the toxic features of trichinosis.

MYIASIS INTESTINALIS.

The larvæ of many different species of flies may live and possibly multiply in the intestinal canal, causing at times grave lesions, ulcerations, perforation, and thickening of the wall of the intestine, with resulting stenosis. The treatment should aim at the early destruction and expulsion of the parasites.

CHAPTER LIII.

DISEASES OF THE RECTUM.

HEMORRHOIDS; TUMORS OF THE RECTUM; STRICTURES OF THE RECTUM; PROCTITIS; ULCERS OF THE RECTUM; PROLAPSE OF THE RECTUM; PROCTOSPASM; PARESIS; COCCYODYNIA.

HEMORRHOIDS.

HEMORRHOIDS, or piles, are either diffuse or circumscribed dilations of the hemorrhoidal veins. The tumors or swellings are situated subcutaneously in the external anal sphincter and submucously in the lowest portion of the rectum. There is often inflammatory infiltration and connective-tissue proliferation. According to the veins affected, a distinction is made between external and internal hemorrhoids.

External hemorrhoids are situated at the anal margin and quite outside the rectum, while internal hemorrhoids are situated entirely inside the rectum and originate from the bowel proper. The distinction between an external and an internal hemorrhoid is not explained by the simple fact that one is below and the other above the external sphincter. A different set of bloodvessels is implicated in each case. An external hemorrhoid is a varicosity of an external hemorrhoidal vein, and is therefore an affection of the general venous circulation. An internal hemorrhoid is a varicosity of the middle or the internal hemorrhoidal vein, both of which are parts of the visceral venous system. A glance at the venous anatomy of the rectum and the anus will show the arrangement of these two sets of veins, and will also explain how, from the free anastomosis which exists between them, it is improbable that one should be affected without influencing the other to a greater or less extent.

Hemorrhoids develop when the flow of blood from the hemorrhoidal veins toward the vena cava and the portal vein is obstructed. The cause of the interference may be situated in the rectum or in some other part of the large intestine. Here, no doubt, chronic constipation and the excessive use of purgatives play an important part. Sedentary habits, so often supposed to be the cause, probably act more indirectly than directly—by inducing constipation. The upright position of man and the absence of valves in the superior mesenteric vein are predisposing causes of a mechanical character. Furthermore, attention should be given to strictures, tumors and foreign bodies in the rectum. Hemorrhoids also make their

ance in cases of chronic colitis and of malignant tumors of the large intestine, especially of the sigmoid flexure. The etiology includes also affections of neighboring organs—bladder, prostate, uterus; pregnancy, if it interferes with the free flow of venous blood by pressure, may be a causative factor. It has not been conclusively proved whether disturbances in the circulation in connection with heart disease may lead directly to the formation of hemorrhoids.

Hemorrhoids may occur in young persons, but the subjects are, as a rule, persons of more mature years. The male sex is decidedly predisposed.

Anatomically, hemorrhoids show themselves either as a diffused tumor surrounding the anus beneath the skin and the mucous membrane, or as circumscribed tumors, single or multiple, of varying size.

Symptoms. Hemorrhoids usually give rise to local symptoms. In some cases, however, large tumors exist without inducing any subjective discomforts. The local manifestations include a feeling of pressure, weight in the rectum, itching, tenesmus, burning and painful sensations in the anus. These symptoms are most strikingly in evidence when the tumors become irritated by inflammation. Hemorrhages are of frequent occurrence, and may be the first symptom to send the patient to the doctor. The symptoms are usually less after an evacuation of the bowels; hemorrhage also has a temporarily relieving effect—evidently because of the abstraction of blood from the hyperemic and inflamed tissues. The hemorrhages are not really beneficial, however, as was formerly supposed, and as is held even today, especially by the laity. The symptoms mentioned above, particularly the pains, may occasionally become so excruciating that the patients contemplate suicide. The complications of hemorrhoids are: catarrhal conditions of the rectum with excessive secretion of mucus, inflammations, excoriations and fissures of the hemorrhoidal nodes. These may give rise to infections and induce abscesses, fistule, and phlegmonous processes. The incarceration of prolapsed hemorrhoids is very painful. When it is impossible to reduce large-sized prolapsed nodules, they may become greatly swollen and then cause the most violent pains (strangulation). Grave anemia may develop in consequence of chronic hemorrhages.

General symptoms caused by hemorrhoids include a feeling of pressure and fulness in the abdomen, sacral and dorsal pains, and nervous phenomena such as pressure in the head, headache, vertigo, and nausea. These symptoms depend upon differences in the relative blood-pressure in the territories drained by the vena cava and the portal vein, chronic constipation, toxemia, and neurasthenia.

Treatment. In the treatment of hemorrhoids the etiology must be taken into consideration; in fact we should always endeavor to remove the cause. Particular attention must be given to the relief

of the chronic constipation which is the most frequent cause of hemorrhoids (see Chapter XXXVII on Chronic Constipation). With respect to diet, a distinction is to be made in the treatment between hemorrhoids with and those without hemorrhage. A careful regulation of the diet is obligatory in cases which are characterized by severe continuous or periodical hemorrhages; alcoholic beverages, sharp spices and highly seasoned food or drink are to be avoided, and the patients must abstain from prolonged walking, climbing, horseback riding, golf, sports, wheeling, and all fatiguing occupations. When the hemorrhages are insignificant or absent, a diet similar to that advised in chronic constipation is to be maintained (see Chapter VII on Diet). An invigorating purgative diet, not too abundant in refuse material, is appropriate in such cases.

Mineral water drinking cures often produce good effects. The sodium chlorid waters are more suitable for lean persons, while the sodium sulphate and the magnesium sulphate waters are more particularly adapted to adipose patients with a certain plethoric habit. The good effect frequently seen after taking these waters is explained by their purgative action. The other favorable factors at a health resort, such as bodily and mental rest, proper diet, frequent baths, and sports, must get some credit for the benefit. Unfortunately the good results of the cure are often transitory, disappearing when the patients return to their homes. In a good many cases, however, an annually repeated course of treatment with such waters is capable of effecting a permanent cure (see page 252).

In every case of hemorrhoids the patients are to be instructed to pay scrupulous attention to the cleansing of the anus. In the case of many patients with hemorrhoids and a sensitive anus, it is not sufficient that the anus be cleaned after defecation with toilet paper alone; it should be washed with warm water held in a piece of absorbent cotton, carefully dried, then bathed in an anti-septic fluid (cold 3-per-cent. boric solution) or an astringent solution (one teaspoonful of tannic acid to one pint of water). After this treatment the anus and the immediate vicinity, especially the tumors, should be coated with a thin layer of petrolatum.

The treatment of *hemorrhage* is of the greatest importance. Severe acute hemorrhage requires tamponing of the rectum. In this work the proctoscope (Fig. 37) is a valuable aid. When the hemorrhages are profuse, hot irrigations of water (95° to 115° F.) act effectively. If necessary, 1- to 2-per-cent. tannic acid may be added. Good styptic effects are also attributed to a 10-per-cent. solution of gelatin in water. A similar effect is produced by the application of wads of cotton saturated in epinephrin solution (1:1000); the epinephrin contracts the bloodvessels markedly. When the seat of the hemorrhage is high up, suppositories containing astringents may be introduced as far as possible. Boas recommends a 10-per-cent. solution of chemically pure chlorid of lime; 20 Cc.

(3v) is injected into the rectum by means of a small syringe twice daily. I have used ten drops of the fluidextractum thuja in a teaspoonful of water to be injected beyond the sphincter, morning and night, by means of a small glass syringe, with benefit.

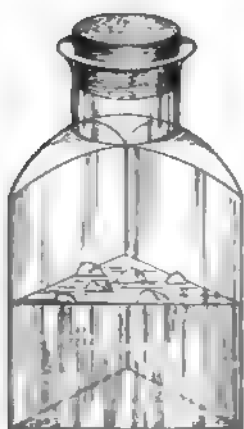


FIG. 145.—Pyramidal form ice-bag.

Should the hemorrhage not be arrested by means of these drugs, anesthesia should be induced and the bleeding points found and ligated or cauterized. Quinin and urea hydrochlorid in 1-per-cent. solution is a very efficient local anesthetic and has been successfully employed in a great variety of operations upon the rectum. It is marketed in hermetically sealed glass ampoules.

For chronic slow hemorrhage, which may in time lead to grave anemia, the administration of hamamelis by mouth may be recommended. Of the fluidextractum hamamelis virginianæ, one teaspoonful is given three times a day, and this medication is continued for months; it is harmless and causes no discomfort. Ergotin and hydrastis

canadensis are much less efficacious (see page 514). An ointment of the following composition may be applied directly to the hemorrhoidal tumors:

	Gm. or Cc.	
R—Extracti hamamelidis virginianæ	0 3	gr. v
Olei theobromatis	10 0	5iiss
Olei amygdalis	7 5	5ij
Misce et ft. unguentum.		
Sig.—Apply		

The pains, which frequently cause great annoyance when the hemorrhoids are inflamed and bleeding, are best treated with rest in bed and cold applications. Zweig has constructed a special ice-bag of pyramidal form (Fig. 145) which, when filled with ice, is to be placed between the gluteal eminences and brought in direct contact with the painful parts; it is retained in position by a T-bandage loosely fastened. For relief of the pains, the following may be applied locally in the form of suppositories or ointments: cocain, eucaïn, morphin, opium, epinephrin, extract of belladonna, eumydrin, anesthesin, orthoform.

	Gr. or Cc.	
R—Anesthesin or orthoform	0 2	gr. iij
Olei theobromatis	2 0	5ss
Misce et ft. suppos. no. 1		
R—Anesthesin or orthoform	0 5	gr. viiss
Aquæ anaë hydrosol.		
Ferr. liq.	33 4 0	5j
Misce et ft. unguentum		

Suppositories of chrysarobin are recommended for their analgesic and styptic effects:

	Gm. or Gs.	
R—Chrysarobin . . .	0.08	gr. 1½
Iodoform	0.02	gr. ½
Extracti belladonnæ	0.01	gr. ¼
Olei theobromatis	2.0	℥ss

Misce et ft. suppos. no. i.

Sig.—One suppository two or three times daily.

	Gm. or Gs.	
R—Chrysarobin	0.08	gr. 1½
Iodoform	0.03	gr. ⅝
Extracti belladonnæ	0.6	gr. x
Petrolati	15.0	℥ss

Misce et ft. unguentum

Sig.—For external hemorrhoids; to be applied several times daily

	Gm. or Gs.	
R—Morphinæ sulphatis	0.015	gr. ¼
Extracti belladonnæ	0.01	gr. ¼
Extracti hyoscyami	0.01	gr. ¼
Olei theobromatis	1.0	gr. xv

Misce et ft. suppos. no. i.

Sig.—Introduce at once, and repeat in two hours if necessary

	Gm. or Gs.	
R—Cocainæ hydrochloridi	0.01	gr. ¼
Extracti opii	0.03	gr. ⅝
Extracti krameriæ	0.5	gr. viiiss
Olei theobromatis	1.0	gr. xv

Misce et ft. suppos. no. i.

Sig.—Introduce at once, and repeat in two hours if necessary

When great pain is experienced, leeches may be placed at some distance from the anus. Ulcerated and gangrenous nodules are to be covered with antiseptic powders. Occasionally it will be necessary to reduce prolapsed nodules which have become incarcerated and cause severe pain. This is best accomplished with the patient in the lateral posture and the pelvis elevated, by pressure with a lubricated wad of gauze and by gentle force with the fingers.



FIG. 149. Dumb-bell pessary

To minimize the pain of this procedure, first apply a 4-per-cent. solution of cocain, or leeches to reduce the congestion. In cases in which the reduced nodules persist in prolapsing, a well oiled dumb-bell pessary (Fig. 149) may be introduced. When the prolapsed nodules are gangrenous they should not be replaced but allowed to become necrotic and fall off, analgesic agents being meanwhile employed. Esmarch's rectal truss (Fig. 150, page 847

of a pea are to be seen, which gradually become progressively paler, to finally disappear altogether. In the first two or three days the patient may have lancinating pains, which can be relieved by the application of aluminum acetate solution or by the use of anodynes. Absolute rest in bed for three or four days is necessary. The bowels should be regulated by mild aperients. It is not necessary to maintain any particular diet. Boas found that the treatment usually covered eight to fourteen days. Ulcerated nodules should be slightly cauterized with silver nitrate or dusted with antiseptic powders. This treatment is indicated above all others in cases in which a radical operation is contra-indicated. It is simple and harmless.

Midway between the radical and the bloodless methods stands the injection treatment of the hemorrhoidal tumors. The author's experience with injection has been satisfactory in a large number of cases, but the method requires a much longer time than surgical operation. Frequently the subsequent removal, under local anesthesia, of leaf-like tags is necessary. In selected cases of non-inflamed internal hemorrhoids, when an anesthetic is contra-indicated or an operation is refused, the injection method has a distinct field. The treatment consists in injecting the nodules with small quantities of a phenol-glycerin solution. For this purpose the nodules are brought before the anus. The anal region is carefully cleansed, the rectum is washed out, and subsequently the anus and the nodules are disinfected with a 0.5-per-cent. solution of lysol. Each nodule is then injected from the periphery, by means of a fine hypodermic needle (Fig. 145), drop by drop, with the phenol-glycerin solution, until the main portion of it turns whitish. A fenestrated rectal speculum (Fig. 152) should be used. If possible the tumors are replaced in the rectum. A large wad of cotton, applied to the anus, is kept in place by a T-bandage. Rest in bed for two or three days is then necessary, with a diet of soups, and opium to prevent movement of the bowels. On the third day castor oil is given. The next few days the patient remains inactive.

The injected hemorrhoid sloughs off. The patient does not know when this occurs, and in normal cases the spot to which it was attached looks healthy and clean. Sometimes a raw surface results, but this soon heals. Should an ulcer develop, due to too large an injection or improper technic, no harm will have been done; treatment as applied to any ulcer will rapidly heal it.

Never inject hemorrhoids that are inflamed or irritated; inject the smaller piles first. Handle all parts with extreme gentleness. Apply alcohol to protect the tissue from the outflow of the phenol solution. Do not inject the second time until all inflammation and soreness has disappeared.

If, upon withdrawal of the needle, blood follows, there has not

been enough of the solution used; reinsert the needle and inject more and allow time for the blood to coagulate. When pain follows the operation, the liberal application of hot water will relieve it.



FIG. 151.—Guarded hypodermic needle for injecting hemorrhoids.

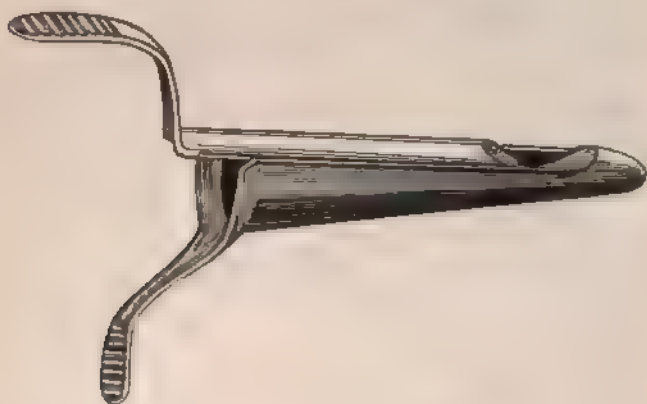


FIG. 152.—Fenestrated rectal speculum.

The following solution may be used:

	Gm. or Gs.	
R Phenolis	45 0	3℥ss
Glycerini	15 0	3ss
Misc.		
Sig. Inject one or more minims		

Quinin and urea hydrochlorid in 5-per-cent. solution has of late been used with marked success. The solution, injected into the hemorrhoid, causes fibrous exudation which restricts the blood supply of the part, with consequent atrophy. One hemorrhoid can be injected every day until all have been treated. If the hemorrhoids are strangulated or greatly inflamed, the patient should be put in bed for a few days and local applications made until the inflammation has subsided, when the injection treatment is begun. The technic in detail as employed by Dr. Terrell is as follows. The hemorrhoids are brought into view through a small conical fenestrated speculum. The hemorrhoid selected for treatment is swabbed with a solution of equal parts of iodine and alcohol. A few drops of a 5-per-cent. solution of quinin and urea hydro-

chlorid are then injected very slowly into the pile at the highest point possible. Just sufficient solution is injected to cause slight distention, a very small needle being used—not large enough to obstruct the view. The needle should be inserted into the body of the hemorrhoid and held in position for a moment after the

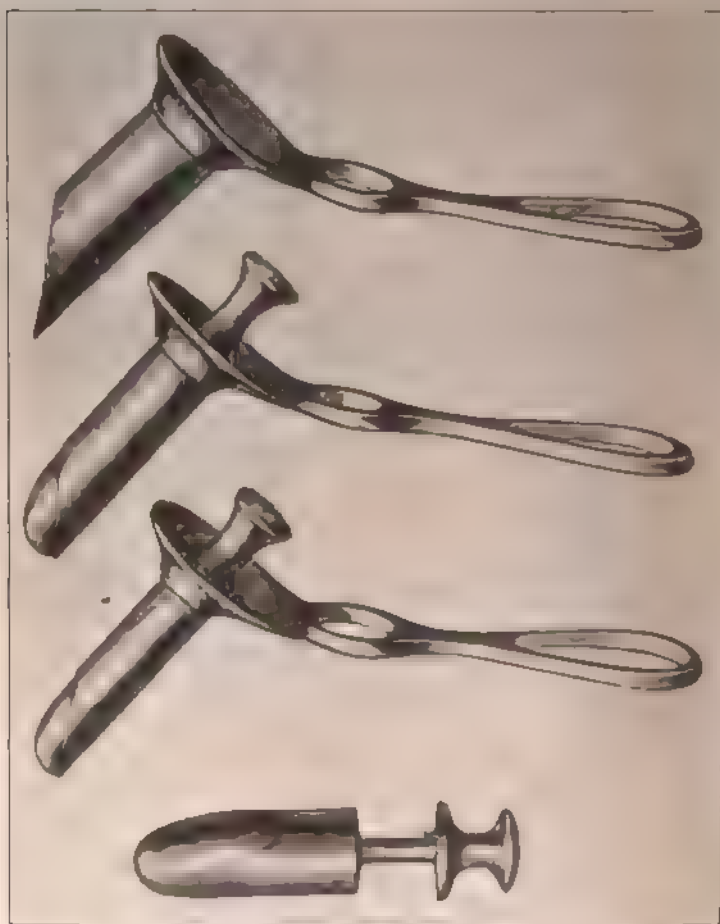


FIG. 153.—Hirschman's anoscope with oblique opening.

injection to prevent bleeding at the point of puncture. The following day, if a digital examination is made, the hemorrhoid will appear thickened and indurated. After a few days it begins to diminish in size, and this diminution continues for ten days to two weeks, the action of the compound seeming to be continuous for that length of time. At the end of the period, if the hemorrhoid

still persists, the treatment should be repeated as before, with a slightly stronger solution.

I find that the Hirschman anoscope (Fig. 153) is the best instrument to use for this treatment. After the anoscope has been introduced and the obturator removed, a slight forward and backward movement of the instrument, under the guidance of the eye, allows the hemorrhoid to fall into the oblique opening. Asking the patient to slightly bear down assists this procedure.

Quinin and urea hydrochlorid is a very decided hemostatic, and a hemorrhoid that has been bleeding freely for some time will rarely bleed after the first treatment. The drug is not an escharotic, but a most excellent local anesthetic. When injected into the hemorrhoid it does not produce an inflammatory reaction or spasm of the sphincters, consequently pain is very seldom complained of. During the progress of these treatments the patient is allowed to continue about his business as usual; no restrictions whatever are placed upon him.

Electrolysis in the treatment of internal hemorrhoids has many advantages over cutting. The technic can be successfully carried out in the office. A single application of the current is effectual in ordinary cases. The patient needs but little preparation before treatment. A cathartic the night before and an enema an hour before coming to the office should be sufficient to prevent any interruption or delay during the treatment. After the usual antiseptic cleansing, the patient is placed in the left lateral position. A short proctoscope is introduced and the hemorrhoid brought into view. It is not necessary to dilate the sphincters. A hypodermic syringe is used to introduce a local anesthetic. Eucain may be used, but the percentage must be very low because the solution does not ooze away as in cutting operations. A larger quantity of fluid is necessary for pressure anesthesia than when injecting into the skin. A solution of 0.1 per cent. is sufficiently concentrated to secure perfect anesthesia. Beta-eucain lactate has the advantage of not being decomposed by boiling, and is said to be less toxic than cocain. An all-metal syringe of two-dram capacity, with a very small needle, is recommended. The hypodermic injection should be made from the base to the apex of the hemorrhoid. When the tissue is distended and has a blanched appearance, the tumor is ready for treatment. Williams¹ recommends a ten-inch monopolar platinum electrode (Fig. 154) having three points arranged in a row. The needles should be passed into the center of the tumor after it is no longer sensitive, and their position changed from time to time until the disintegration of the tumor is complete. This pole is attached to a negative electrode. The

¹ *Electricity in Rectal Diseases*, New York Medical Journal, April 26, 1913.

current should never be interrupted. The rheostat should always be turned to zero. A current of two to twenty milliamperes slowly turned on and continued five to ten minutes is usually sufficient to treat a single hemorrhoid. As soon as the current is turned on, bubbles of gas form in the shrinking tumor, giving it a grayish-white appearance. The chemical effect is disintegration of the albuminous constituents. It is not necessary for the patient to remain in bed. When there are several hemorrhoids, they should be treated ten days to two weeks apart. There is no hemorrhage during or after the treatment.

The surgical operations for hemorrhoids are: ligature, the Whitehead operation and its modifications, and the clamp and cautery. The reader is referred to works on surgery for detailed descriptions of these operations.

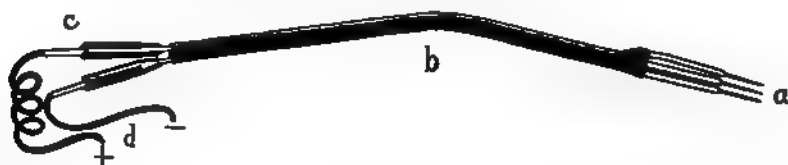


FIG. 154.—Hemorrhoidal electrode. (Williams.) *a*, needles; *b*, electrode; *c*, electric connection; *d*, electric wires.

MALIGNANT GROWTHS.

Carcinoma.—It has been mentioned in the paragraph on Tumors of the Intestine that the great majority, 70 to 80 per cent., of all the cases of intestinal carcinoma are carcinomata of the rectum. The development of a rectal carcinoma is very insidious. The first symptoms are usually connected with a history of retardation in the evacuation of the bowel, the course of which resembles that of an ordinary chronic constipation. The constipation is usually treated by purgatives. Gradually, however, the irregularity in the bowel movements assumes more striking features. The patients are obliged to go frequently to stool, but, notwithstanding great efforts, only small quantities of fragmentary fecal masses are expelled. The evacuation at this stage approximates in appearance the so-called fragmentary stool of Boas (see page 672). Defecation is not followed by the usual relief. (Plate XXVIII.)

Symptoms.—One symptom which Leaf always regards with suspicion is the constant passage of flatus through the anal orifice. I have been able to verify the value of this symptom in the detection of the growth.

The patients complain of pressure and fulness in the rectum and a backache in the sacral or coccygeal region. Pains of various

PLATE XXVIII



**Carcinoma of the Rectum with Dilatation and Ulceration
of the Sigmoid Flexure.**

degrees gradually appear. The appetite diminishes and the patients become pale and emaciated. The stools become soft, then liquid, then decomposed; when examined carefully, blood, mucus and pus are demonstrable. By digital exploration the carcinoma is discovered situated at a variable distance from the anus. It presents itself either as a spacious cavity with rigid, ragged, rough walls (especially in cases of deep-seated carcinomata), or as an isolated annular tumor with so small an aperture as scarcely to permit the insertion of a finger (especially when the carcinoma is located high up).

The cylindric or columnar carcinoma is the variety that most frequently involves the rectum. It feels like a raised, warty growth, embedded in the rectal walls (Leaf).

Diagnosis.—To firmly establish the diagnosis, it is important to ascertain how far the carcinoma extends upward; this is accomplished by digital examination or by the use of the proctoscope. The Roentgen ray is of great assistance (Plate XXII, Fig. 2). Other important features are, the mobility of the tumor, and the firmness with which it has become attached to the periproctal tissues. Metastases are most frequently found in the liver.

The complications of rectal carcinoma are, invasion of neighboring organs (bladder, genitals), and penetration of the skin of the anal region. Perirectal (peri-anal) abscesses are rare. The differential diagnosis between carcinoma and syphilitic stricture of the rectum occasionally offers difficulties; the Wassermann reaction is a valuable aid. The serologic tests for carcinoma are helpful (see page 543).

Treatment.—Carcinomata of the rectum are the most favorable of all the intestinal cancers in respect to treatment, because it is possible to make an early diagnosis and the opportunity is thus afforded to radically remove them by surgical intervention. In every case of carcinoma of the rectum, therefore, the first consideration is the feasibility of operative removal. Generally speaking, those carcinomata are considered operable which are movable. The degree of the extension and involvement of the carcinoma is not of so much importance, so far as operation is concerned, as fixation. The general condition of the patient is of greatest importance; operation is frequently inopportune because of marked cachexia and anemia.

Many permanent recoveries have been obtained by operation. Recurrences and metastases, particularly in the liver, are very frequent; these complications soon terminate fatally, even after a favorable result of the operation *per se*. With regard to the functional effects of the operation, it is of great importance to preserve the sphincter if possible; but even so, rectal function is scarcely ever as perfect as before the operation. When it is impossible to preserve the sphincter, the patients are unable to retain either feces or flatus.

Inoperable carcinoma of the rectum is, characteristically, elongated, ulcerated, ragged in outline, and firmly embedded in and involving the surrounding structures. The question arises, whether in such a case an artificial anus should be established before severe symptoms of stricture develop. Many surgeons defer the operation of colostomy until the development of symptoms of ileus, and avoid the artificial anus as long as the lumen remains tolerably patent. There can be no doubt that for many sensitive patients an artificial anus is absolutely intolerable. Suicide under such conditions is not rare. An indifferent patient, on the contrary, may frequently be able to accommodate himself fairly well to the artificial anus. Here, therefore, the question of the individual should be taken into consideration. A colostomy is indicated in some cases for the reason that by diverting the course of the feces it allows perfect quiescence of the carcinoma, and this may result in an abatement of the inflammatory processes, with cessation of hemorrhage and ulcerative discharge, and the conversion of an inoperable carcinoma into an operable one.

Adenocarcinoma responds well to radium, the squamous-celled variety not so readily.

If radical or palliative operative procedures are out of the question, symptomatic treatment is only possible by means of internal medication. The strength of the patient should be maintained as much as possible by proper food, rich in calories; a strictly liquid diet is seldom necessary. Moreover, the stools should be kept as soft and semiliquid as possible in order to avoid stagnation from hard fecal masses above the carcinoma. This is best accomplished by the administration of purgative foods (see page 182) and mild laxatives.

Great benefit can frequently be derived from liquid petrolatum in 30-Gm. (3j) doses three times a day. The dose can usually be taken with a pinch of salt, if at first disliked. The oil lubricates the mucous membrane and the feces and renders the passage of the latter less irritating to the ulcerated neoplasm (see page 664).

Pains and tenesmus are to be combated by opium, morphin, codein, and belladonna, by mouth or subcutaneously, and occasionally in suppository form. Anodynes should be used freely (page 274).

In the presence of markedly fetid discharges, hemorrhages, and purulent disintegration, attempts may be made to counteract these complications by irrigation of the rectum with antiseptic liquids or by means of the dry treatment through the proctoscope with the powders previously named (see page 237). The results thus obtained are occasionally quite satisfactory.

Sarcoma.—Sarcomata of the intestine are most frequent in the small intestine and the rectum. The clinical symptoms of sarcoma of the rectum are, in general, identical with those of carcinoma,

the only difference being the more rapid course of the sarcoma, due to its quicker growth. Sarcoma makes its appearance earlier in life than rectal carcinoma. The surface of the sarcomatous tumor is smooth to the touch, and the tumor does not show so marked a tendency to disintegration as does carcinoma.

Treatment. The treatment is similar to that of carcinoma of the rectum.

BENIGN GROWTHS.

Polypi. Polypi of the intestine are classed in pathologic anatomy as adenomata, and are situated on a broad base or connected by a narrow pedicle to the mucous membrane. They may occur singly, but are usually found in groups. Their size varies from that of a pea to that of an egg. Though no part of the intestine is immune, their point of predilection is the rectum. A single polypus, or even a small group, may exist without producing any symptoms. Minute hemorrhages are sometimes due to this cause. But when one or several large polypi are present in the rectum, disagreeable symptoms follow, and profuse bleeding may occur; pains develop, and when the polypi are very large they narrow the intestinal lumen considerably or may even obstruct it entirely. Rectal polypi can be diagnosed by digital exploration or by means of the proctoscope. They can be distinguished from malignant neoplasms by their complete isolation, by the pedicle, and by the absence of ulceration and cachexia.

Treatment. Polypi in the rectum should be removed radically, for the reason that they are apt, under certain conditions, to undergo malignant degeneration. When it is possible to bring deep-seated polypi out of the anus, they should be ligated and cut off. Polypi situated farther up, especially those with pedicles, may be removed through the proctoscope with a long-handled sharp spoon or by means of a snare similar to that employed for the removal of small polypi of the nose. A polypus may be seized with a sharp hooked forceps and removed by torsion or by careful avulsion. The latter procedure is easy, and it often happens that pedunculated polypi fall off spontaneously. Polypi may likewise be extirpated at their base by means of the electric cautery snare. The remaining stump of the pedicle of the polypus, after removal of the latter, is slightly cauterized with nitrate of silver in order to prevent hemorrhage. The stumps of the larger sized adenomata and polypi may be destroyed by means of intrarectal Roentgen-ray treatment. Some cases of multiple polyposis are benefited by radium.

The prognosis assumes a much more serious aspect when, instead of a few polypi or a single isolated one, the entire rectum is closely studded with large and small polypi—a condition which is designated

as *polyposis recti*. These cases are serious for the reason that they are very prone to undergo carcinomatous degeneration and give rise to grave chronic hemorrhages; every defecation is accompanied by the spontaneous discharge of blood, serum, pus, and mucus, frequently in such volume as to gradually bring about great emaciation and anemia. These conditions are often accompanied by severe pains and tenesmus. The effects of internal and surgical treatment are satisfactory. Internally hemostatic medicaments (ergotin, hydrastis canadensis, hamamelis) may be administered. The local treatment consists of irrigations with astringent and hemostatic drugs, such as tannic acid, alum, and gelatin. A few of the larger nodules should be removed, and this operation is occasionally followed by improvement of the entire process. The establishment of an artificial anus sometimes acts beneficially.

The *polyposis recti* is occasionally accompanied by polyposis of the entire large intestine. In some few cases a family predisposition to polyposis has been noted.

Lipomata and Myomata.—Both these affections are very rare. Lipomata originate in the submucous coat, and their favorite seat is the rectum or the large intestine; they may attain considerable magnitude (size of a child's head), and they occur either singly or in groups. Myomata develop from the mucous or submucous coats (internal myoma), or from the subserous coat (external myoma); they are found more frequently in the colon than in the rectum; no age is exempt; the tumors sometimes attain to the size of a man's head.

Internal myomata frequently invest the mucous membrane in the form of pedunculated tumors and thus give the impression of ordinary polypi; under such circumstances it is not easy to make a differential diagnosis. Clinically they are marked by fecal urgency, the passage of blood and mucus, and by the signs of rectal stenosis.

The symptoms of external myomata are less characteristic. These growths may possibly form adhesions with the pelvic organs, thus causing pressure and inducing rectal bleeding.

Treatment.—The treatment of lipomata and myomata may be expectant, so long as no urgent symptoms are manifest. Spontaneous expulsion of the tumor has, though rarely, been observed. Otherwise only the radical operation is indicated. Internal pedunculated myomata are treated similarly to polypi. External myomata are to be operated upon like carcinoma of the rectum.

Papillæ. Papillæ are small nipple-shaped elevations, frequently found just above Hilton's white line. They are of elongated conical shape, and may undergo mucoid degeneration. They are the cause of pruritus and many neurotic symptoms and are highly sensitive to pressure.

Treatment.—Their treatment consists in radical removal from the mucous membrane at their base.

STRICTURES OF THE RECTUM.

Strictures of the rectum may develop in consequence of diseases located in the neighborhood of the rectum, or from a diseased condition of the rectum itself. Narrowing from the outside may be induced by tumors, exudates, stones in the bladder, or hypertrophy of the prostate. Internal strictures may be caused by fecal lumps, enteroliths, tumors of the rectum, or local inflammatory affections (Plate XXII, Fig. 3).

All diseases of the rectum which heal with the formation of cicatrices may induce narrowing of the rectal lumen. This is the case with rectal ulcers, and among these, in direct order of frequency, are the syphilitic and the dysenteric ulcers. There is also a purely inflammatory form of rectal stenosis, the genesis of which resembles that of chronic inflammatory hypertrophy of the pylorus; to chronic proctitis is added loss of mucous membrane, and round-cell infiltration with proliferation of the interstitial tissue. Most cases of rectal stenosis are in women who have or have had syphilis. The strictures are, as a rule, situated near the anus, rarely higher up; there is a more or less extensive stricture, formed by tough masses of connective tissue, which is sometimes associated with ulceration. Occasionally the entire rectum becomes converted into a rigid tube. Not infrequently several strictures are present simultaneously.

Symptoms.—Patients with stricture of the rectum usually neglect the slight initial symptoms, and only when the discomforts become more urgent do they seek medical advice. The pathologic nature of the stricture determines the symptoms, such as excretion of blood, pus, mucus, liquid stools, or evacuation of firm fecal fragments of small size alternating with diarrhea. All this is accompanied by pain and tenesmus which may become excruciating. Occasionally the sphincter becomes destroyed, with resulting incontinence of feces.

Diagnosis. The stricture is diagnosed by digital examination; if high up, by the use of sounds or the proctoscope. Pus and blood in the feces and the shape of the stools assist in the diagnosis. It is, however, often extremely difficult to recognize strictures located high up.

Rarely there occurs a congenital stricture of the rectum; the diagnosis is readily made by digital examination.

The nature of the stricture often remains undetermined. The distinction between syphilitic and dysenteric strictures, particularly, offers great difficulties. The most important distinction to be

made is that between malignant and non-malignant. In a general way Tuttle¹ distinguishes them as follows:

MALIGNANT STRICTURE.

Generally occurs in persons above thirty-five years of age.

Runs its course ordinarily in two or three years. Constitutional symptoms, such as loss of flesh and strength, appear early in the course of the disease.

Hereditary influence probable.

To the touch, hard, nodular, without pedicle; protrudes into the rectum from more or less of the circumference of the gut, but not equally; it may occur as a deep excavating ulcer with sharp edges and indurated base, or sometimes as a fungous, granulating, cauliflower growth. May be movable, but is usually attached to the sacrum and surrounding parts.

The odor is nauseating, gangrenous, and unique.

NON-MALIGNANT STRICTURE.

Occurs at any age, ordinarily between twenty and fifty.

The patients may live for many years with it. General health remains good through long periods.

No hereditary connections.

To the touch it is smooth, hard, and inelastic, but not nodular. A distinct cicatricial or fibrous appearance upon examination through the speculum.

Rarely attached to the sacrum, but sometimes attached to organs in the anterior portion of the pelvis.

The odor is fecal or feculent, according to the amount of ulceration.

The discharge may be abundant or limited, thick or thin, according to the nature of the stricture.

Treatment. The treatment by internal medication is by no means successful in cases of rectal stricture. When syphilis is suspected, antisyphilitic treatment should be instituted at once, although the results are, unfortunately, often negative. Mild aperients are necessary, in order to alleviate, to a degree at least, the very harassing symptoms of stenosis (see page 285).



FIG. 133. Dilator.

The medical and surgical treatment of strictures is of great importance and interest to physicians and surgeons and stretching of the contracture. According to the reports of various authors, the results of the various procedures are not uniform. It must, however, be remembered that the success of the treatment depends upon the nature of the stricture and the skill of the operator and patient. Stages of various degrees of severity may be met with. As the stretching must be done with great care and patience, it is not possible to give a definite statement of the results of the various procedures.

¹See Tuttle's "Diseases of the Rectum," 1887, p. 100.

soft-rubber hollow bougies containing a metal spiral to stiffen them. Soft-rubber Wales bougies (Fig. 105) are the best instruments for this purpose. With great caution, hard-rubber bougies may be used; they are either straight or curved, as Cr  d  's instrument (Fig. 155). Care must always be exercised lest the bougie pass through the rectal wall instead of the stenosed canal.

Dudley Roberts describes an apparatus he uses for gradual anal dilatation (Figs. 156 and 157). To an inner bag of rubberized cloth, the ends made bulbous to prevent slipping inward or outward when distended, is attached a tube of like material, on the end of which is fastened a small stopcock; a hand bulb, valved



FIG. 156.—Rectal dilator deflated.
(Roberts.)

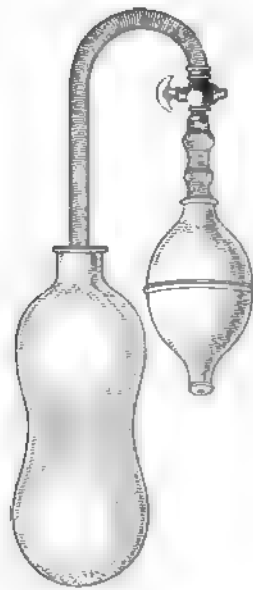


FIG. 157.—Rectal dilator inflated.
(Roberts.)

to prevent the backward passage of air, is attached to the stopcock. Within the bag and extending through a portion of the tube is a slender metal rod with bulbous ends; this is a simple means of giving the collapsed bag sufficient rigidity during introduction. Outside the strong dilating bag is a thin elastic cover free from seams, which gives a perfect smoothness to the bag at all stages of dilatation. The method of use is exceedingly simple, and few directions are necessary. The bag is well dusted with talcum powder or covered with an emollient; the elastic cover is then slipped on and moved around to completely lubricate apposed surfaces. Two-thirds of the length of the bag is introduced, through an anal speculum, and slow dilatation is started. As soon as discomfort is felt the stopcock is turned and a few minutes are allowed to elapse

in order that the voluntary and involuntary spasm may be relaxed. Gradually the dilatation is continued, and when as much as possible has been done the bag is left in place for ten to fifteen minutes. The patient is instructed to lie flat on his back and relax completely. Successive treatments follow, and each time dilatation is found to be easier until a normal condition is established. The advantage



FIG. 158.—Rectal dilator, closed. (Rosenberg.)

of this form of instrument in the treatment of strictures of the rectum above the anus is obvious.

Other useful instruments are sea-tangle tents, and bougies with olive-pointed ends similar to the esophageal sound with an olive point. Whitehead has described an apparatus made of rubber which is introduced deflated into the stricture and afterward

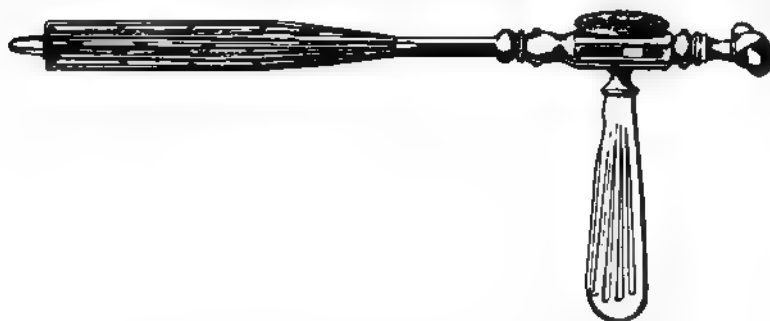


FIG. 159.—Rectal dilator, open (Rosenberg.)

distended with water. Dilatation with these instruments is to take place daily or every two or three days, at first for a few minutes, and later gradually prolonged to an hour or more. By careful torsion of the bougie the stretching effect may be enhanced. In order to obtain the best results it is necessary to continue the treatment for months, with a constant increase in the caliber of the sounds, and to repeat the process from time to time.

Rosenberg recently devised an instrument for stretching strictures of the rectum (Figs. 158 and 159). It is constructed on the same principle as the urethral dilators; apart from the size, it differs in construction and in its great strength, making dilatation possible even when the strictures are very firm and hard. The branches open by the twisting of the handle, and a scale permits exact reading of the degree of the dilatation. After the location and the width of the stenosis have been ascertained, the apparatus, closed and covered with a condom, is introduced into the stricture. The branches are then opened carefully a millimeter at a time, by turning the lever, until resistance is encountered; the stretching is then continued with the greatest caution, the operator constantly observing the scale of the apparatus and the face of the patient. When the patient experiences great tension or pain, the stretching is discontinued and the apparatus is left *in situ* for twenty to thirty minutes. Should it then be possible to dilate further, this is accomplished most carefully. When removing the apparatus the branches should not be put into juxtaposition, for fear of catching and compressing a fold of the mucous membrane. It is said that with this method the patient experiences only a feeling of tension, never violent pain; the irritation of the cicatrix and mucous membrane is reduced to a minimum, and the degree of dilatation is absolutely within the hands of the physician. Therefore improvement or cure results much more rapidly than with the other methods of dilatation.

When these bloodless methods do not accomplish their purpose it becomes necessary to resort to surgery.

PROCTITIS.

Primary inflammation of the mucous membrane of the rectum is induced principally by mechanical, chemical or bacterial irritants, which find their way into the rectum either with the feces or from the outside. Secondary proctitis is found associated with other diseases of the rectum. Primary proctitis occurs in both acute and chronic forms, and may remain circumscribed or become diffused over the entire surface of the rectum.

Symptoms. Acute proctitis is accompanied by sensations of pressure, tension, and irritating itching. When the inflammation is extensive it is accompanied by pains and tenesmus, which sometimes become quite severe. Defecation under such circumstances is difficult. Mucus, blood and pus are excreted with the feces. The general health is considerably affected, and fever may be present.

Diagnosis. The diagnosis is easily established by the aid of the proctoscope (Fig. 37). Chronic proctitis is usually a secondary condition and is found associated with fissures, strictures, hemor-

rhoids, and foreign bodies in the rectum; the subjective sensations are usually less pronounced than those of acute proctitis.

Treatment.—Should the cause of acute proctitis be found (foreign bodies, hard scybala, affections of neighboring organs), it must of course receive proper attention. Complete bodily rest is essential. The patient should be placed in bed, and the rectum kept quiescent by the administration of opium by mouth or by suppository. Cool, moist applications of alum or fuller's earth, or an ice-bag, are placed over the perineum. Leeches may be applied in the vicinity of the anus. The diet should consist entirely of liquids. Lying on the abdomen or on one side often lessens the pains. With absolute rest the inflammatory symptoms soon recede. Activity of the bowels may be kept up by the administration of castor oil or by enemata of olive oil. Direct local treatment in acute proctitis is not indicated. When the more intense inflammatory irritation has passed off, the mucous membrane may be irrigated with chamomile tea, linseed tea, demulcents, and highly diluted astringents; if necessary, a few drops of tincture of opium may be added. Acute gonorrheal proctitis is treated locally with antigonorrheal remedies, such as protargol and albargin in 0.25 to 1 per cent. solution injected into the rectum and retained as long as thirty minutes.

In chronic proctitis the bowels are to be regulated, and the rectum is to be washed out once or twice daily with the usual astringent and antiseptic solutions. Irrigation of the mucous membrane of the rectum with hot water (110° to 120° F.) is valuable. For this purpose the irrigating instrument (Fig. 44) should be used. When the instrument has been introduced into the rectum it should remain there until the seance is finished; if removed before all the hot water has come away, it may burn the skin. It must be remembered that the rectum can stand water at a higher temperature than the skin. Many medicated solutions can be used for the irrigation: phenol 0.5 to 1 per cent., sodium salicylate 1 per cent., thymol 2 per cent., potassium chlorate 1 per cent., boric acid 1 per cent., hydrastis 1 per cent., and aqueous fluid extract of *krameria* 5 to 20 per cent. When a decided astringent is necessary, nitrate of silver 1 per cent. or tannic acid 3 per cent. in distilled water can be used. Certain oils, as cedar, cajuput, or spruce, may also be used, being mixed with the water or solutions by the addition of magnesium carbonate. While they will not really mix with the water, these oils will be so well subdivided that they will find their way into the rectum and act as stimulants, deodorants, and antiseptics (Albright).

Hot water or hot medicated solutions are essential to the successful treatment of this condition, the heat being probably of more value than the medication. Nothing excels hot water for rectal pain, either by irrigation or applied externally by means of the sitz bath (see page 250).

Astringent and antiseptic remedies may also be applied in the form of suppositories, or introduced into the rectum directly in the form of semiliquid salves by means of an ointment syringe; or the salves may be expressed from the original tin tubes connected with a piece of soft-rubber tubing. Rodari obtained good results from the application of a tannin-ichthyol solution followed by silver nitrate suppositories. In the morning, if possible after defecation, he orders the injection into the rectum of the following solution, which is to be retained as long as possible:

	Gm. or Cc.	
R—Acidi tannici	0.5	gr viiss
Ichthyoli	6.0	ʒiss
Alcoholis	10.0	ʒiiss
Aquæ destillatæ q. s. ad	100.0	ʒiij
Misce.		
Sig.—To be injected.		

In the evening, previous to retiring, a suppository of the following composition is introduced:

	Gm. or Cc.	
R—Argenti nitratis	0.03	gr. ss
Olei theobromatis	3.0	gr. xlv
Misce et ft. suppo., no. i.		

Sitz baths and local astringents assist these curative measures. Sea baths are also said to act beneficially. The drinking cures of Saratoga and Carlsbad and the bitter mineral waters are valuable (see page 252).

The cases that require operation are those in which there is hypertrophy of the mucous membrane and enlargement of the rectal valves (hypertrophic proctitis). In such cases the redundant tissue is pushed down in front of the descending feces and forms a temporary obstruction at Houston's valves. Division of the rectal valves gives more room for the feces, and this operation may occasionally be necessary to overcome the rectal constipation (Plate XX, Figs. 3 and 4).

ULCERS OF THE RECTUM.

All varieties of ulcers may occur in the rectum, just as in the large and small intestine, as discussed in detail in the chapters on Intestinal Ulcers. They are tubercular, dysenteric, follicular, and stercoral, and the ulcers that are found in connection with chronic ulcerative colitis. Syphilitic and gonorrheal ulcers are frequently observed in the rectum, although they are rarely found in other parts of the intestinal tract. Syphilitic ulcers in the rectum develop from disintegrating broad condylomata or from necrotic gummata. They are usually situated low down in the rectum, and sometimes destroy the sphincter. The soft chancre may also be localized about the anus. Gonorrheal ulcers subsequent to infection of the mucous membrane with gonococci

are usually located on the anterior and posterior walls of the anal orifice. Traumatic ulcers may easily result from the careless application of enemata.

Diagnosis.—The diagnosis of ulcers is made with the aid of digital and proctoscopic examinations, the study of the feces for blood, pus, tubercle bacilli, and endamebæ, examination of the genitals, previous history, and study of the clinical condition, particularly tuberculosis of the lungs.

Treatment.—In cases in which the ulcers are due to syphilis or tuberculosis, the cause must be properly treated. Tubercular ulcers offer little hope in this respect, since tuberculosis of the rectum is practically always complicated with tubercular infection of the large or small intestine. In syphilitic ulcers, on the contrary, especially if they are of rather recent origin, specific treatment by arsphenamine, mercury and the iodids often effects a cure. Recovery is much more difficult and uncertain when the specific ulcers are of long standing, with large ragged surfaces that have caused inflammatory infiltrations in the neighborhood. In such cases, even should the treatment prove successful, the hard cicatricial tissue present is apt to lead to stricture of the rectum. The simple primary sore heals in the same manner as in other locations. In gonorrheal ulcerations, if the patient is a woman the genitals should be carefully treated in order to prevent reinfection of the intestinal mucous membrane.

Besides the general treatment, local treatment of the ulcers must be considered. The dysenteric, tubercular, stercoral and follicular ulcers are treated by either the moist or the dry method, as described on page 250. In syphilitic and gonorrheal ulcers the diet should be carefully regulated in such a manner as to avoid constipation. The direct treatment consists of irrigation of the rectal mucous membrane with astringent and antiseptic solutions, as silver nitrate, sulphate of zinc, tannin, and alum, or insufflation of antiseptic powders through the proctoscope. Cauterization with chlorid of zinc or phenol may be performed through the proctoscope. The above-named medicaments may also be introduced into the rectum in the form of thin fluid ointments by means of an ointment syringe. To alleviate the pains, anodynes, such as anesthesin and orthoform, may be added to these ointments.

	Gm. or Ce.	
R—Orthoformi	5	0
Adipis læs hydrosi, Petrolati	āā	12 0
Misce et ft. unguentum.		
Sig.—To be introduced with an ointment syringe twice daily.		

	Gm. or Ce.	
R—Anesthesini, Bismuthi subgallatis	āā	3 0
Petrolati	30	0
Misce et ft. unguentum.		
Sig.—Apply with a syringe twice a day.		

gr. xiv
5j

When the pains are severe the following astringent ointment can be used:

	Gm. or Oz.	
R—Morphine sulphatis	0 1	gr. ij
Unguenti plumbi,		
Petrolati	℥ 10 0	℥ iiss
Misce et ft. unguentum.		

Price has had good results from the following:

	Gm. or Oz.	
R—Extracti hamamelidis destillatæ	200 0	℥ viij
Extracti hydrastidis (aqueous)	10 0	℥ iiss
Phenolis	2 0	℥ xxx
Glycerini	35 0	℥ ix
Misce		

Sig—Mix one-half teaspoonful of this mixture with one-half teaspoonful of corn starch and two tablespoonfuls of warm water. Inject into the rectum with a hard-rubber syringe and retain all night.

Irrigations of the rectum with antiseptic and astringent solutions, as described in discussing the treatment of proctitis, are sometimes valuable.

In severe cases these methods of treatment occasionally fail, because recesses and stenoses are present which prevent the access of the medicaments to the diseased parts. In such cases surgical treatment becomes necessary. Sometimes a thorough curettement of the ulcers, followed by the actual cautery, and division of the sphincter, will effect a cure. In order to induce drainage, sphincterotomy has been proposed. In ulcers located high up, colostomy and local treatment through the artificial opening should be kept in mind. In extensive ulcerative degeneration it sometimes becomes necessary to resect the rectum.

PROLAPSE OF THE RECTUM.

(*Procidentia Recti*.)

Prolapse of the rectum is seen most frequently in infancy, particularly during the second and third years of life; it may be induced by whooping-cough, emaciation, constipation, intestinal catarrh, rectal catarrh, or tenesmus. Prolapse is rather rare in adults. The same causes as in children act similarly; and besides, hemorrhoids, diseases of the bladder and the sexual organs, pregnancy, and senile atrophy of the pelvic muscles may induce prolapse. The development of this displacement proceeds slowly: at first merely the anal part of the rectum protrudes during defecation, and later a large portion of the rectum. When the prolapse is extensive, Douglas's pouch will contain intestinal loops. This condition is termed *hernia recti*.

Prolapses of large size which do not return spontaneously give rise to a great deal of discomfort. The mechanical irritation of the

exposed mucous membrane induces inflammation, catarrh, and the discharge of mucus. Moreover, hemorrhages, erosions and ulcerations may develop. To this are added irritating pains, quite apart from the constant annoyance caused by the soiling of the clothing and the fetid odor which interferes with all the pleasure of life and diminishes all social interest. When the prolapse becomes more chronic, the inflammatory symptoms generally recede and the mucous membrane becomes more skin-like and less sensitive. In recent cases the prolapse may become incarcerated and gangrenous.

Treatment.—Internal treatment during the initial stage promises good results. Rather small and recent cases of prolapse may be cured by regulation of the bowels, avoidance of strong bearing-down pressure, tonic treatment of the rectum such as cold washing and sitz baths, with astringent solutions, douches, and, in children, painting of the mucous membrane with 1- to 5-per-cent. solutions of silver nitrate.

Treatment in cases of infantile prolapse demands, according to Milward:

- (a) Removal of the exciting cause.
- (b) Measures for the relief of the predisposing causes.
- (c) Reduction and maintenance in position of the displaced gut.
- (d) Vesical calculus, phimosis, worms, diarrhea, constipation, etc., must be efficiently treated.

(b) Nutrition must be improved by correcting the diet, and debility met by the exhibition of tonics. These children are often underfed or ill-nourished by insufficiency of milk and the substitution of artificial foods. In the way of tonics they may be given iron, the syrup of hypophosphites, malt extract, and cod-liver oil.

(c) The reduction of the prolapse is effected by washing it with cold water, gently squeezing out the edema with the flat of the four fingers and the thumb, and pressing it back inside the anus between the expiratory gasps. The fingers should be well oiled, and the child's buttocks slightly raised. When the prolapse is quite reduced, a small pad of folded lint is put over the anus, and the buttocks are strapped together over it. Several strips of adhesive plaster are necessary, and the physician should finally see that the reduction is maintained—that the rectal mucous membrane does not appear at the edge of the straps. It is also well to bandage the thighs together.

In adults subcutaneous injections of ergotin 0.1 to 0.2 Gm. 2 to 3 grains and strychnin 0.001 to 0.002 $\frac{1}{2}$ to $\frac{1}{3}$ grain may be made into the region. These injections are said to be often efficacious in strengthening the muscular power of the sphincter. Similar good results sometimes follow lavage of the rectum and intrarectal faradization. Massage of the sphincter is said to powerfully strengthen the muscular tonus. After reducing the

displaced gut, circular movements are performed with the index finger outside of the sphincter ani, kneading of the muscles of the floor of the pelvis, and finally vibrations with the finger introduced into the rectum. When massage is practiced in this manner once or twice daily, fairly good closure of the sphincter may be brought about.

Non-replaceable prolapse should be prevented from protruding by a properly adjusted rectal truss. Such an apparatus as the rectal truss of Esmarch (Fig. 160) is of value. The method of application is sufficiently shown by the illustration. This apparatus is made as a firm oval bath of rubber about the length of a finger, which adapts itself to the size and situation of the rectum. In children, one of the best methods of keeping up the prolapsed rectum is to strap the buttocks together after each defecation. It is occasionally possible to retain mild prolapses by cotton wads and a T-bandage.



FIG. 160 Rectal truss (Esmarch).

A recent prolapse may usually be reduced by gentle pressure of the finger with the aid of an oiled compress. When a prolapse has existed for some time or when it is incarcerated, it may become very difficult of reduction. The patient is then placed in the left lateral position, with the pelvis elevated, and attempts are made to replace the prolapse by gentle massage. Should this be unsuccessful, the prolapse may be gradually reduced by a cotton wad and a T-bandage. Sometimes it is necessary to give a general anesthetic or to induce anesthesia of the parts by the intraspinal method in order to reduce the prolapse.

Hajeck recommends the following treatment for rectal prolapse in children: A tapering piece of ice, about three inches long, and one inch in diameter at the large end, is wrapped with iodotorn gauze, and its point pressed gently against the center of the prolapsed mass until the latter is replaced; the ice tampon will remain in the rectum without the help of any retentive bandage, provided it is pushed in far enough. A fresh piece of ice is employed in this way after each act of defecation. This treatment soon cures the prolapse; it seems to act by emptying the bloodvessels and heightening the contractility of the rectum.

The indication for surgical intervention depends on the degree of discomfort experienced. In cases in which there are permanent

marked disturbances, pains and hemorrhages, and in which the above internal and mechanical procedures do not lead to success, operative measures should be resorted to. Operations for prolapse are, as a rule, not particularly dangerous.

PROCTOSPASM.

The tendency of the intestine to contract spasmodically increases with the distance the contents have traveled: the severe spasms occur mostly in the rectum and anus. Proctospasm leads to irregular evacuation and constipation, particularly when a spastic contraction of the anal muscles accompanies it. The spasm is usually followed by a very unpleasant, even painful pressure of desire for stool, without result (see page 388).

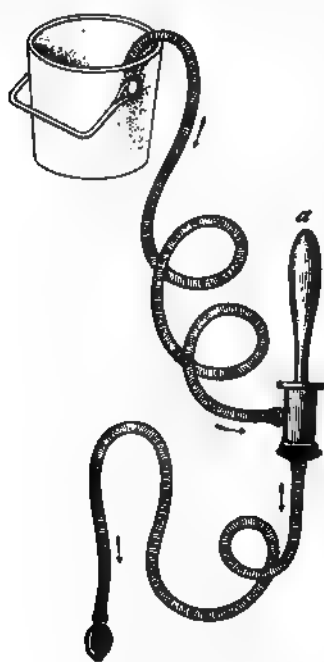


FIG. 161.—Rectal refrigerator.
(Ataperger)

The symptoms of proctospasm are violent pains in the rectum, with a persistent continuous contraction of the anal sphincter. The pains occur usually at night in periodic seizures; the patient awakens with the pain, which may attain great intensity and is characterized by the sensation of spasmodic anal contraction. This is what is actually taking place. If the attempt be made to insert the finger into the anus, this will be found to be scarcely possible. The attack usually persists for some minutes.

The tendency toward constriction of the lower sections of the intestine, easily recognizable roentgenographically, usually develops secondarily from local affections of the rectum and the anus—inflammations, ulcers, fissures, hemorrhoids, and foreign bodies

—or from inflammation of the pelvic organs in the female or cystitis and affections of the prostate in the male. Proctospasm arises also through irritation of the vegetative nervous system of the pelvic area, *e. g.*, in crises of tabes, hysteria, and neurasthenia. Tenesmus with intense pains, which increase paroxysmally up to attacks of weakness, occurs during the passage of the stool, and continues for some time after; it may even extend to the bladder and induce ischuria. The diagnosis is made by digital examination by way of the rectum, or by roentgenologic examination after an enema of bismuth emulsion.

Treatment.—In cases of purely functional proctospasm the treatment must be directed to the general nervous disease underlying the spasm. The local measures especially to be considered are: galvanization of the abdomen and rectum with weak currents, dilatation of the anus with rectal sounds of increasing sizes, and application of the rectal refrigerator. Atzperger's apparatus (Fig. 161) is a useful instrument for this purpose. This rectal refrigerator consists of a hollow metal cone (*a*) about seven centimeters ($2\frac{1}{2}$ inches) long, furnished with a contrivance for the circulation of cold water. The end of the instrument is thoroughly lubricated and inserted into the rectum, and water of the desired temperature is allowed to circulate. Occasionally hot water (100° F.) acts beneficially. Internally the bromids should be given or attempts should be made to prevent the development of the attacks by the administration of opium, morphin, belladonna, codein, or cocain, by mouth or as suppositories (see page 658). Should there be spastic constipation (see Chapter XXXVII), it must be carefully treated. Papaverin or benzyl benzoate will relieve the spasm (see page 276).

PARESIS AND PARALYSIS OF THE RECTUM.

Paresis and paralysis of the rectum are very rare as primary nervous conditions. They are most frequent as a result of local rectal affections (prolapse, tumors, proctitis, hemorrhoids), diseases of the genitals (hypertrophy of the prostate, strictures of the urethra), chronic constipation, and organic diseases of the brain and spinal cord. The diagnosis is made by inserting the finger and ascertaining the absolute relaxation and inability of the sphincter ani to close. Patients are unable to retain the feces during bodily exertion, coughing or sneezing. In mild cases involuntary defecation takes place only with diarrhea, but in severe cases formed fecal matter is discharged involuntarily.

Treatment.—The causative disease, whatever it may be, must be properly treated. If none is discoverable, the treatment should be directed toward improvement of the function of the sphincter. This is primarily attained by faradization of the rectum, continued regularly for a long time. The diet must be arranged as indicated by the condition of the stools. Diarrhea must be treated with antidiarrheic remedies. Should the feces be very firm, they should be rendered softer by means of the diet, because then the evacuation of the bowel is more complete and the involuntary escape of feces is restricted. The patient should carefully regulate the time of taking food and, as far as possible, of evacuation of the bowels. Should masses of feces be firmly lodged in the ampulla recti and remain stationary there, they must be softened by irrigation. The sphincter ani may be rendered more contractile by cold sitz baths,

cold ablutions of the perineum, and cold douches of the rectum. Strychnin may be given subcutaneously, 0.001 to 0.002 Gm. ($\frac{1}{80}$ to $\frac{1}{40}$ grain), or in suppositories as extract of nux vomica 0.03 Gm. ($\frac{1}{4}$ grain), twice daily (see Chapter XI).

COCCYGODYNIA.

Coccygodynia, or pain in the region of the coccyx, occurs in women, and is increased by walking, micturition, and defecation. Another affection which may simulate rectal neuralgia is the anal crisis of tabes.

Treatment.—Ely has suggested a treatment for coccygodynia which I have found highly satisfactory. It consists of massage of the coccyx by means of the forefinger in the rectum and the thumb on the outside, holding the bone between them. The bone is moved backward and forward, and the soft parts are moved about on the bone. The manipulation is begun very lightly, and gradually increased in force as the patient becomes less sensitive. Usually a few treatments at intervals of two or three days will suffice for a cure. The improvement is almost immediate.

It is observed that a sedative ointment rubbed into the skin of the coccygeal region sometimes gives relief. The following ointment is often found to be efficacious:

	Gm. or Cc.	
R—Tincture aconiti	2 0	ʒss
Unguenti belladonnae	30 0	ʒj
Misce et fit unguentum.		

Sig.—Apply thoroughly.

Alcohol induces degeneration of sensory nerves and is useful in cases of coccygodynia. Ten to twenty minims of 80-per-cent. alcohol are slowly injected at the point of pain. With the right index finger in the rectum, the maximum tenderness is determined by counter-pressure with the thumb on the outside. The finger in the rectum acts as a guide to the needle, directing it to the painful spot, at the same time guarding against perforation. The pain from the puncture lasts a few minutes and is followed by a dull ache. Four or five injections at intervals of one week are necessary.

The application of the actual cautery is said to constitute the most certain remedy. A Paquelin cautery is used to cauterize the skin over the sacral foramina on each side, the skin being burnt deeply, and the resulting eschar is then treated as an ordinary granulating wound. Coccygectomy may be necessary.

CHAPTER LIV.

DISEASES OF THE ANUS.

PRURITUS ANI; ANAL FISTULA; FISSURE OF THE ANUS.

PRURITUS ANI.

PAINFUL itching in the anal region is called pruritus ani. This itching is merely a symptom and not a rectal disease *per se*. There are many causes of this distressing affection: constipation, hemorrhoids, fissure, fistula, ulcer, proctitis, polypi, papillæ, inverted hairs, jaundice, gout, carcinoma, syphilis, foreign bodies, diabetes, uterine diseases. Pediculi and ascariæ are frequently associated with pruritus ani. Wallis believes the pruritus is due to a small ulcer located between the two sphincters. He says that in over 90 per cent. of the cases so examined a shallow ulcer was found, usually between the two sphincters, more often in the posterior half than in the anterior, and generally in the dorsal midline; in some cases there is more than one ulcer, and again in others there are various clefts which occasionally almost surround the bowel. The ulcer is not easy to recognize by the touch, and it requires a certain amount of practice to appreciate its presence. In the first place it must be remembered that it is only just within the anal margin and always below the internal sphincter. The smooth feeling of the healthy lining membrane will be recognized, but when the finger comes to this abraded or ulcerated surface the smooth feeling disappears and a slightly raised margin can be felt around the rough surface. There is sometimes pain, but more often none, associated with the examination. When the speculum is introduced it must be remembered that the tissues are pushed in, some little distance, by the instrument, and so the ulcer will appear to be higher up than it really is. If after its introduction the speculum be opened to its fullest extent, the inexperienced observer will probably not recognize the ulcer; but if it be only slightly opened and a careful view with a headlight obtained, the ulcer can be clearly seen as a shallow, oval, livid abrasion, differing markedly, and mainly in color, from the normal mucous membrane. Here, then, is a definite lesion in a so far indefinite disease, and it has seemed reasonable to believe that it might be the cause of the irritation.

According to Beach, the distinct pathogenesis of pruritus ani consists of single or multiple burrowings from the anal pockets,

emitting a serous or seropurulent substance; the sinus may be complete or blind and is always accompanied by proctitis, and frequently by cryptitis and small ulcers at the anorectal line. There is occasionally a causal relationship between tabes and pruritus. This possibility should be kept in mind.

Many cases of pruritus ani are due to infection by the *Streptococcus fecalis*, the portal of entry being the anal canal.

Pruritus ani essentialis is due not to a local but to a constitutional cause. It is a primary affection, conditioned upon trophic changes in the nerves supplying the parts. In examining a well developed and well established case of this kind it is noticed that the skin of the anus and immediate vicinity has become thickened, is drier than normal, and has lost its pliability; that it is raised into numerous radiating folds from which the normal coloring matter of the skin is absent; it presents a grayish, parchment-like appearance. All these changes in the condition of the skin can be readily accounted for if we assume that the primary cause of the trouble is a faulty nerve supply of the parts. The loss of pigment is due to absorption of the coloring matter of the deeper layer of the skin, induced not by irritation of the parts from scratching, but by abnormal innervation. The appearance of the skin is quite typical and should be recognized as pathognomonic. The chronic inflammation of the epidermis completely alters its character; it becomes largely infiltrated with fibrous tissue, loses its elasticity, and becomes covered with dead and sodden epithelium (Wallis).

The skin in pruritus ani may be either moist or dry. The dry, scaly type is usually found in neurotic subjects, while the moist variety is found in the plethoric. The anal zone is eczematous for the space of perhaps an inch on every side, or it may be well over the buttocks, the eczema being the result of scratching or of serous discharges from the anal canal.

The following, according to Leaf, are the local and general conditions usually found associated with pruritus:

LOCAL.	GENERAL.
1. Disease of the colon, rectum, or anus, especially fissure, ulcer, chronic ulceration, polypi, piles, prolapse of mucous membrane, fistula, condylomata, malignant growths.	1. Irregularities in diet. Consumption of shellfish, lobster, or salmon. Drinking tea, coffee, cocoa, or beer.
2. Skin affections of the anogluteal region, erythema, eczema, tinea circinata caused by the trichophyton.	2. Constipation. Negligence in attending to nature's calls.
3. Thread worms.	3. Gout, diabetes, Bright's disease, rheumatism, syphilis.
4. Diseases of neighboring organs—uterus, ovaries, bladder, prostate, etc.	4. Overwork.
5. Uncleanliness.	5. Irritable or neurotic temperament.

The itching may become intolerable, especially in the warmth of the bed. Not only children, but adults also, are apt to scratch the itching parts until open sores result.

Treatment.—The radical treatment consists in removal of the cause. Constipation must always receive proper treatment (see Chapter XXXVII on Chronic Constipation). The stagnation of feces produces venous congestion, which superinduces excretion of mucus, thus keeping the anus moist and provoking dermatitis. The same thing occurs in hemorrhoids, fissure, fistula, ulcers, proctitis, prolapse, and neoplasms. It is of the utmost importance that the anal region be kept clean. Fecal soiling together with slight moisture may cause intolerable pruritus. The use of printed paper and coarse toilet paper for the toilet must be prohibited. Pediculi and worms must receive due attention. This is important in the pruritus ani of children, for intestinal worms are often present (see page 804).

Any general disease implicated must have proper consideration; this includes the treatment of diabetes, gout, rheumatism, cholecystitis with obstructive jaundice, and uremia. Highly seasoned food, lobster, crab, and strong tea or coffee must be interdicted, as well as all alcoholic drinks and tobacco. An enlarged prostate or a displaced uterus must be corrected. Vaginal discharges require local treatment. Primary skin diseases, such as eczema, herpes, and ringworm, must receive proper attention.

While treating the underlying cause, some direct relief of the itching must be effected if possible. Goodell recommends the following soothing application:

	Gm. or Gr	
R Chloral hydrate,		
Camphor	aa	15 0
M et adde		
Acid borici,		
Unguenti simplex	aa	15 0
Miscer		
		3ss

Sig.—Apply with a brush three times daily, after cleansing the parts with hot water.

It is sometimes advisable to add to the above ointment 1 to 1½ grams (15 to 20 grains) of phenol, and in other instances 1 to 1½ grams of menthol. Applied at bedtime this will often insure a good night's rest.

Compound tincture of benzoin is a mild styptic and antiseptic. It can be applied to the anal region every night. The balsam causes all the small cracks to heal. The application must be continued for several weeks. If the parts are moist they may be dusted with starch, calomel, bismuth subnitrate, zinc oxid, boric acid, prepared chalk, sulphur, or Bulkley's antipruritic powder—which is prepared by rubbing together 4 Gm. (5j) each of camphor and chloral until liquefied, and adding this to 30 Gm. (3j) of starch.

Long persisting anal pruritus can frequently be relieved by rubbing the region with dry calomel. The part is first wiped dry with absorbent cotton, and, with a moistened finger cot over the index finger, the powder is rubbed thoroughly into the crevices. After four or five applications the itching usually subsides permanently.

Daily ablution of the anus with hot water and soap followed by friction of the anal region with a 70-per-cent. alcohol and 1:1000 sublimate solution will often afford relief. The following mixture is very efficacious:

		Gm. or Gr.	
R	Glycerini,		
	Alcoholis	53	60 0
	Acidi salicylici . . .	2	0
Misce,			3ij 3ss

Sig. To be applied several times daily after a hot ablution of the anal region.

Should the itching interfere with sleep, the local application of hot water for fifteen to twenty minutes at bedtime will usually procure a night's rest for the patient.



FIG. 162. Anal ointment introducer.

Citrine ointment (U. S. P.) is frequently useful. It can be applied through the ointment introducer or pile pipe (Fig. 162). As a soothing, non-irritant application the resorcinol ointment of the *National Formulary* can be used with the pile pipe.

The following is soothing and is said to give almost immediate relief:

	Gm. or Cc	gr. xx
R Phenolis	1.25	
Hydrargyri chloridi mitis,		
Picis	55	4 0
Mentholis	0 1	3j
Zinci oxidi	4 0	gr ij
Petrolati	24 0	5j
Adipis lane hydrae	8 0	5vj
Misce et ft. unguentum		5ij

Sig = Bathe with water as hot as can be borne and apply the ointment twice daily

The following has also been found very beneficial:

	Gm. or Cc	
R Anesthesini	4 0	3j
Zinci oxidi	2 0	3ss
Adipis lane hydrae	15 0	3ss
Petrolati	15 0	3ss
Misce et ft. unguentum		

Sig = Apply three times daily

Pruritus being a result of pressure upon the nerve terminals by increased blood supply on the one hand and a scaly or horny condition of the skin on the other, the chief indication is to diminish the blood supply and desensitize the nerve endings, and this indication is fully met by the hypodermic injection of quinin and urea hydrochlorid solution. The needle is inserted about an inch from the anal margin, on a line with the tuberosity of the ischium, and a 0.5 per-cent. solution of the double salt directed first toward the back of the anus, then laterally, and again, with a guiding finger in the anus, well toward the perineal raphe. Thus three injections are made from one point of insertion (about 30 minims being introduced at each injection), and this operation is repeated on the other side of the anus. The liquid is placed as near as possible to the anal mucosa, but the sphincter must be avoided. The relief afforded by this procedure lasts from two to four weeks.

Gratifying results are often obtained from the use of anal dilators. The patient, immediately after retiring, inserts into the anus as large a dilator as it will comfortably accommodate, and allows it to remain at least fifteen minutes. Let him do this every night until the muscles have relaxed sufficiently to receive a larger size. This treatment should be continued, as in other instances of gradual dilatation, until the sphincter is more relaxed than it is intended ultimately to be. This method, unlike divulsion, does not injure the mucous membrane. The small, blunt, hard-rubber instruments found in the shops in graduated sizes are all that is necessary; and they are inexpensive (Fig. 52). The treatment is to be followed by extreme dilatation with the Roberts rectal dilator (Figs. 156 and 157).

Allingham has used an anal plug (Figs. 163 and 164) to prevent nocturnal itching. The plug exercises pressure upon the venous plexuses and filaments of nerves close to the anus. It was suggested to him by patients telling him they could obtain relief and sleep by introducing the end of the forefinger into the anus and making pressure that instantly arrested the irritation.

In exceptionally violent cases where local applications give no relief the Roentgen ray may have the desired effect. Two or three applications at three-week intervals have been found to be effectual. Relief usually comes fifteen days after the first application. The cases which are the most benefited by the Roentgen ray are those in which lichenification has set in, with or without an eczematous condition and oozing.



FIG. 163. —Anal plug.

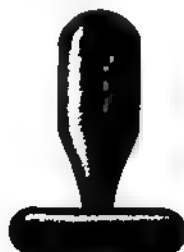


FIG. 164. —Anal plug.

Pruritus ani is an affection which one would not think of referring to bacterial infection. Wright says that it had not occurred to him that it might be due to such a cause until a patient who was suffering from this condition was referred to him for the treatment of an associated furunculosis. He now finds it difficult to understand how it is possible to look at pruritus ani from any other point of view than that of a bacterial infection. He has found in a number of cases that a platinum loop applied to the seat of irritation brought away quite astonishing numbers of bacteria, invariably staphylococci and pseudodiphtheria bacilli, and occasionally tetrads; and in each of these cases life has been rendered comfortable or, if not quite comfortable, at any rate quite endurable, by the use of appropriate bacterial vaccines. Murray had cultures made in 98 cases of pruritus ani, and found *Streptococcus fecalis* present externally on the skin in 85 of the cases. A blood test in each of the 85 cases further showed that the patient's resistance against streptococci was low, while it remained high for other microorganisms. All of these cases were treated with autogenous vaccine, with the result that itching ceased and streptococci ceased to be found in smear and swab after a period of treatment varying from the first to the eighth injection. In one case a lowered resistance to the *Bacillus coli* was also found, and in this case vaccines of both organisms were used.

Should these methods fail, it is frequently imperative to resort to surgical measures. It may be necessary to remove hypertrophied skin folds. Anal pockets must be opened and drained. Anal papillæ are to be removed. The operation devised by Ball has given the best results; it consists in dividing all the sensory nerves supplying the skin of the anus and anal canal which proceed from branches of the third and fourth sacral nerves. This operation renders the region superficially anesthetic, and the pruritus is immediately relieved.

ANAL FISTULA.

A fistula begins in an abscess in the cellular tissue around the anus. This abscess may be directly under the skin or in the submucous tissue. Instead of forming an exit, the pus may burrow in many directions, making honeycomb ramifications. The abscess may break through the outer skin or through the mucous membrane, making a sinus. The longer a fistula is left, the more does it burrow and the more difficult it is to cure.

A simple fistula, as a rule, extends from a point between the internal and external sphincters a distance of about one and a half inches from the anus. The ends of the fistulous tract are usually small openings, while the space between may be long and tortuous. The internal opening may be high up in the rectum or quite low. The fistulous tract is usually lined with a pyogenic membrane, and pus exudes from all parts of its ramifications. The classification of fistulæ depends upon the situation of their openings.

A complete fistula opens on both the mucous membrane and the cutaneous surface. A complete external fistula has both openings on the cutaneous surface. A complete internal fistula has both openings on the mucous membrane. A blind external fistula has one opening only—on the cutaneous surface. A blind internal fistula opens on the mucous membrane only.

A variety of complete fistula called the "horseshoe" runs from one ischiorectal fossa to the other, usually with one external and one or two internal openings.

Whether the opening be internal only or the fistula be complete, the watery and acrid pus which contaminates the anal margin is apt to set up very intense pruritus, and thus fistula is a very common cause of this latter condition. The itching in these cases is also said to be due to the presence of minute fecal particles caught in the granulations of the fistulous opening, as is the case in anal fissure.

Fistulæ frequently follow proctitis, ulcers, hemorrhoids, and strictures. The inflammatory process penetrates into the periproctal tissues, inducing infiltration, and this becomes infected so that an abscess develops. This abscess may burrow toward the rectum or under the external integument of the anal region, perforating and thus establishing a fistula. When a periproctal abscess rup-

tures into the rectum only, but not through the skin, the condition is termed an incomplete internal fistulæ. Tubercular fistulæ are very frequent.

Diagnosis.—The most reliable diagnostic measure to determine the existence of a complete fistula is to inject peroxid of hydrogen into the external opening. If the fistula is incomplete, tension and pain will follow; if it is complete, foam will escape from the anus. The internal opening in a complete fistula is a matter of very considerable importance, as, unless acquainted with its usual position (between the sphincters), the surgeon may fail to find it. Bismuth paste or carmin injected in the outer opening of a complete fistula can be easily seen coming through the internal opening (see page 128).

Under certain circumstances fistulæ may develop without the knowledge of the patient, whose attention is aroused only when the fistula, external or internal, discharges purulent matter which moistens and soils the linen. The personal discomforts caused by these conditions may be slight at the time. On the other hand, very considerable inflammatory phenomena and severe pain may be induced by fistulæ retaining their contents. Probing the fistula will demonstrate the diagnosis and its anatomic nature (complete or incomplete fistula).

Treatment.—The treatment of fistula belongs entirely to the surgeon. Antiseptic irrigations rarely cure. Allingham used phenol with some success. Rose completely cured a rectal fistula with the use of carbon dioxid. A current of the gas was passed into the external opening, through the sinus and into the rectum, filling the bowel to its full capacity. The fistula was completely and entirely closed and healed after twelve treatments.

Pennington reports having treated many cases with Beck's bismuth paste. In most of these the fistulæ were located in the posterior anal quadrant, and apparently were not very extensive. All of them, however, communicated with the rectum. The injections were given once or twice a week, and the treatment extended over a period of two to six weeks. The apparent results were eminently satisfactory.

Complete fistulæ should be opened along their entire extent and packed. Incomplete fistulæ are first converted into complete ones and then incised. Abscesses in the vicinity of the rectum require surgical intervention. Pains and inflammatory conditions are to be treated by rest in bed, anodynes, ice, and sitz baths.

FISSURE OF THE ANUS.

Fissures of the anus are small or large elongated oval tears and linear ulcers at the margin of the anus. They usually consist of a solution in the continuity of the mucous membrane, with a yellowish-gray base, and secrete scarcely any noticeable pus. Only when

they have existed for some time and attained considerable depth are they covered with a mucoid exudate. The linear ulcers occasionally penetrate deep into the muscle and expose the fibers of the external sphincter. Anal fissures may be very long and spread as far up as the mucous membrane of the rectum; they are usually situated in the median line, in either the anterior or the posterior commissure, very rarely laterally.

The external end of the fissure is occasionally the site of a small fleshy prominence or "sentinel pile," the explanation of which we owe to Ball. He has clearly shown that this is an anal valve of Morgagni which has been dragged down by the pressure of hardened feces and has left in its track a longitudinal ulcer or fissure extending from the level of the normal ring of valves to the outside of the anus, where fecal pressure can no longer exert its influence. In such cases the sentinel pile will always point to this particular type of fissure and denote the true nature of its origin. Fissures occasionally give rise to and become complicated with blind internal fistulae, due to burrowing from the ulcerated surface. The diagnosis of this condition must be made by observing the exudation of a definite amount of pus and by the use of a probe.

The cause of these tears cannot always be elicited. Chronic constipation with very hard feces contributes to their development, especially when the anus is very tight and the anal skin tender. They may also be caused mechanically, especially from the use of toilet paper. Fissures make their appearance as secondary ulcers in consequence of gonorrhea, syphilis, tuberculosis, and hemorrhoids. They occur more frequently in the female than in the male.

Symptoms. Clinically, anal fissure is characterized first by pains, which are particularly marked during the passage of feces through the torn anus. Severe pain frequently occurs in the fissure even after defecation, evoking reflex spasms. The pains may radiate toward the bladder, the genitals, and the thighs. Even with minute fissures the pain is often intolerable. Hemorrhage from the fissure is frequent during defecation. The patients are inconvenienced extremely by these pains; they anxiously refrain from going to stool, and even avoid the expulsion of flatus. The symptoms are least apparent during bodily rest. Pruritus is a common symptom of fissure and may be the only symptom which induces the patient to seek advice.

The diagnosis is easily made by a careful inspection of the anus. It is, however, essential to smooth out the anus entirely, as small fissures are often deeply concealed in the folds at the mucocutaneous junction.

Treatment. A good deal can be accomplished by prophylaxis in persons who have previously been afflicted with fissures. Appropriate treatment of the chronic constipation and extreme cleanliness of the anus will do much as preventive measures. Such persons should avoid the exertion of much pressure during defecation.

To bring about the healing of a recent fissure as quickly as possible it is best to place the patient in bed for at least one week, retard the defecation artificially by prescribing a liquid diet, and administer ten drops of the tincture of opium three times a day. The fissure itself should be covered with a dusting powder (xeroform, iodoform, anesthesin, orthoform, chlorethane), and all unnecessary manipulations of the anus avoided. Eight days later a large dose of castor oil is given. The patients should go to stool only when they have the sensation that the fecal matter has become liquid. To effect a complete cure, it is important that the passage of feces be suspended for fully eight days. Frequently, however, this object is not attained in eight days, and in such cases the treatment must be repeated once or twice.

An alternative method, recommended by a number of authors, seems to be quite successful. Here defecation is not interrupted; on the contrary, it is increased by a chemically active diet and by mild purgatives (castor oil, mineral salts, bitter saline waters) or oil enemata ($\frac{1}{2}$ pint at night). At the same time rest in bed, if possible, is very beneficial. The patient during this time may be on full diet. Before every defecation a small wad of absorbent cotton saturated with a 5- to 10-per-cent. solution of cocain is inserted into the rectum; the cotton remains there, to be expelled painlessly with the pending stool. The care of the anus must be very thorough after every act of defecation, in order to avoid stretching or irritation of the fissure. One of the best applications is a suppository of opium 0.02 Gm. ($\frac{1}{2}$ grain) and extract of belladonna 0.01 Gm. ($\frac{1}{2}$ grain). By these methods it is possible to bring about painless evacuations and to avoid unnecessary irritation of the fissure, thus accelerating the process of healing.

But in case of failure frequent cauterization with pure silver nitrate, 10-per-cent. nitrate-of-silver solution, or pure ichthyol is beneficial. Cauterization should never be practiced without the previous induction of local anesthesia. The fissure can be easily anesthetized by means of a wad of absorbent cotton saturated with a 2- to 5-per-cent. solution of cocain and allowed to remain in the anus a few minutes. Without local anesthesia intolerable pains follow the cauterization. The fissure, after cocainization, may be energetically brushed twice daily with pure ichthyol; or it can be first dusted with anesthesin or orthoform and then painted with ichthyol. This is to be done daily for one week and then every other day until the ulcer is healed. If there has been much spasm of the sphincter, Tuttle smears the parts with an ointment compound, as follows:

	Gm. or Gs.	
R—Unguenti stramonii,		
Unguenti belladonnae,		
Unguenti hyoscyami	ss 15 0	3iv
Misce et ft. unguentum.		
Sig.—Apply freely.		

This has always seemed to relieve the spasm, and controls the pain resulting from the application of the ichthyol. When the fissures are small, a camel-hair brush saturated with pure ichthyol may be introduced into the anus without cocain; the anus then contracts reflexly and presses the ichthyol into all its recesses and folds. Instead of this, when the patients are very sensitive, an ichthyol ointment has been found useful:

	Gm. or Cc.	
R—Cocainæ hydrochloridi,		
Extracti belladonnæ	ss	0 06 gr. j
Ichthyoli	6 0	3iss
Misce.		

Sig.—To be introduced warm by means of a sound wrapped with absorbent cotton.

A superficial cauterization may also be induced by a Paquelin thermocautery; it is essential, however, to cocainize previously and to apply afterward the following ointment:

	Gm. or Cc.	
R—Acidi borici	3 0	gr. xlv
Cocainæ	1 0	gr. xv
Adipis lanæ hydnosi	25 0	3vj
Petrolati	6 0	3iss
Misce et ft. unguentum.		
Sig.—Apply.		

The entire region of the anus must be repeatedly covered with this ointment.

For hemorrhage, epinephrin can be used. Epinephrin suppositories have a cooling, hemostatic effect, and can be employed with gratifying results. Epinephrin ointment also is available.

Cool sitz baths and local applications are valuable adjuvants to the treatment of the fissure.

It is found that the wearing of a hemorrhoidal pessary for ten to fourteen days (daily one or two hours) is capable of relieving the pains and will often bring about a cure (Figs. 149 and 150).

Doctor Bensaude, of Paris, highly recommends the high-frequency current in every case of fissure. He introduces the electrode into the rectum and allows the current to pass through for five minutes at each seance. The patient receives a treatment every day until the pain is relieved and then every other day. Recovery usually takes place in two to three weeks.

Dilatation or divulsion of the anal sphincters often brings about complete recovery. This operation may be done with a bivalve speculum, but better with the fingers. The two first fingers are gently inserted into the rectum after the patient has been fully anesthetized. The anus is then thoroughly stretched, antero-posteriorly first, then laterally. This is repeated until all spasm has

disappeared and the canal remains patulous. The operation must not be overdone, or it may lead to permanent relaxation.

Should these methods fail to bring about a recovery, surgical intervention is necessary. Surgical methods of treatment consist in the superficial and deep section of the fissure, or completely cutting the external sphincter muscle in such a way that its fibers are divided transversely.

COMPARATIVE SCALES OF THE METRIC AND APOTHECARIES' WEIGHTS AND MEASURES.

Gm. or Cc.	Fluid Measure.			Apothecaries' Weight.	
	Ounces.	Minims.		Ounces.	Grains.
1000	=	33 +	390.6	=	32 + 72.4
500	=	16 +	435.3	=	16 + 36.2
250	=	8 +	217.7	=	8 + 18.1
100	=	3 +	183.1	=	3 + 103.2
50	=	1 +	331.5	=	1 + 291.6
25	=		405.77	=	385.8
10	=		162.31	=	154.3
5	=		81.16	=	77.2
1	=		16.23	=	15.4

Fluid Measure.		Metric.	
2 pints	=	946.358	Gm. or Cc.
1 pint	=	473.179	Gm. or Cc.
$\frac{1}{2}$ pint	=	236.590	Gm. or Cc.
3 ounces	=	88.721	Gm. or Cc.
2 ounces	=	59.147	Gm. or Cc.
1 ounce	=	29.573	Gm. or Cc.
60 minims	=	3.697	Gm. or Cc.

INDEX.

A

- AARON'S abdominal bandage, 575
 - improved stomach tube, 68-70
 - sign of chronic appendicitis, 773
 - of gastroenteroptosis, 565
 - stomach tube and bulb, 69
- Abderhalden reaction in gastric carcinoma, 544
- Abdominal angina from arteriosclerosis, 526
 - bandage, Aaron's, 575
 - Rose's adhesive, 578
 - bandages, 575-578
 - indications for, 575, 605
 - belt, 578
 - corsets, 580
 - massage, 212, 214
- Abscess of lips, 334
 - retropharyngeal, 348
 - of tongue, 324
 - tubercular, of pharynx, 348
- Absorption of aqueous and saline solutions, 61
 - of carbohydrate, 61
 - of cellulose and hemicellulose, 61
 - of fat, 60
 - in large intestine, 61
 - of protein, 60
 - in small intestine, 60
- Absorptive power of stomach, 56
 - Günzburg's method of testing, 89
- Acetylsalicylic acid, occasional oral effects of, 301
- Achalasia, 394
- Achlorhydria, carbohydrate digestion in, 85
 - hemorrhagica gastrica, 464
- Achroodextrin, 50
- Achylia gastrica, 95, 464
 - carcinoma and, 464
 - cholelithiasis and, 464
 - congenital, 465
 - connective tissue in stools of, 674
 - etiology of, 465
 - pathology of, 465
 - secondary, 466
 - senilis, 464
 - stomach contents in, 95
 - symptoms of, 465
 - test-diet stool findings in, 131
- Achylia gastrica, treatment of, dietary, 466
 - by lavage, 471
 - medicinal, 469
 - antiseptic, 470
 - hydrochloric acid, 469
 - pancreatin, 469
 - papain, 469
 - stomachics, 469
 - physical, 472
 - with mineral waters, 472
- Acid secretion after milk, meat and bread, 53
- Acidity of filtered and unfiltered gastric contents, 78
 - of stomach contents, Benedict's effervescence test for, 88
 - dimethylamidoazobenzol test for, 75
 - Friedrich's test for, 89
 - gauze test for, 89
 - gelatin test for, 84
 - Günzburg's tests, 76, 89
 - phenolphthalein test, 79
 - quantitative test, 89
 - Sahl's desmoid test, 90
 - thread test for, 88
 - Töpfer's test (quantitative), 80
- Acidol in gastric diseases, 260
- Acidosis in typhoid fever, 716
- Acids in hyperchlorhydria, 432
- Acorn cocoa, 194
- Actinomyces of esophagus, 361
 - in man, 316
 - of salivary glands, 329
 - treatment of, 316
- Activators of digestion, 53
- Adamantoma of maxilla, 345
- Adenocarcinoma of stomach, 540
- Adenoma, 555
 - of mouth, 340
- Adhesions, gastric massage for, 208
 - intestinal, Roentgen fluoroscopy in diagnosis of, 148
- Adrenalin (epinephrin) in anal fissure, 861
 - in bleeding hemorrhoids, 823
 - in diagnosis of pancreatic insufficiency, 626
 - in esophageal corrosions, 358
 - in dysenteric pains, 725, 726, 727

- Adrenalin in gastric hemorrhage, 271,
519
irrigations in dysentery, 726
in nervous diarrhea, 682
in pylorospasm, 411
in vomiting of pregnancy, 404
- Aerophagy, 401
diagnosis of, 402
flatulence and, 701
hyperalimentation in, 402
psychic treatment of, 402
stomach tube in, 402
- Agar in constipation, 184, 662
tannin, 278
tubes for estimation of duodenal
enzymes, 102
- Akerhelm's rectal friction treatment
of chronic constipation, 229
- Akoria, 416
- Albuminuria in acute intestinal catarrh,
636
- Albumoses, 54
- Alcohol content of artificial foods, 189,
190
effect of, on gastric digestion, 168
secretion, 267
in food cures, 572
- Aleurum flour, 191
- Alimentary catarrh, 635
hypersecretion, 447
- Alimentation, duodenal, 500 *See also*
Diet and Hyperalimentation.
in esophageal stricture, 372
in gastric ulcer, 500
in hepatic cirrhosis, 594
- Alzaria in computing combined HCl,
81
- Alkali carbonated waters, 253
carbonates, 264
chlorin waters, 252
earths, 264
poisoning, fatality of, 357
- Alkalis and alkaloids in hyperchlorhy-
dria, 436, 437, 438
in gastric disease, 263, 265
- Alveolar processes, affections of, 331
sarcoma, 346
- Amelie dysentery, emetin in treatment
of, 723
emetin-mouth iodid in treat-
ment of, 724
- Aminocids, 10
- Ampoules of aseptic solutions, 581
- Amyl nitrite as a gastric sedative, 268
- Amylolectin, 50
- Amylopsin, 58
- Anal dilatation in rectal stricture, 539
in spastic constipation, 672
fissure, 565
cauterization of, 560, 561
dilatation in, 560
diagnosis of, 559
diet in, 560
dilatation of sphincters in, 561
electricity in, 561
- Anal fissure, epinephrin in, 861
etiology of, 859
hemorrhage of, 861
hemorrhoidal pessaries in, 861
ichthyol in, 860, 861
"sentinel pile" of, 859
silver nitrate in, 860
sphincter spasm in, 860
surgery in, 862
symptoms of, 859
treatment of, 859
- fistula, 857
antecedents of, 857
bismuth paste in, 858
complete, 857
diagnosis of, 858
"horseshoe," 857
hydrogen peroxid in, 858
simple, 857
surgery of, 858
symptoms of, 858
- plugs, 856
refrigerator, 824
- Analysis, fractional, of gastric secretion,
78
quantitative, of stomach contents,
76
- Anastalsis of intestine, 63
- Anemia, ground itch, 811, 813
from hemorrhoids, 822
of hookworm disease, 811
in intestinal toxemia, 689
miner's, 806
pernicious, sore mouth in, 302
urobilin and urobilinogen in, 110
- Anesthesia as a gastric anodyne, 270
- Aneurysm, racemose, of mouth, 336
- Angina, abdominal, 523
Ludwig's, 350
- Angioma, cavernous, of mouth, 336
- Anguillula infection, symptoms of, 816
treatment of, 816
intestinalis, 815
- Animal parasites in esophagus, 361
in intestine, 794
in mouth, 320
protein preparations, 187
- Ankylostoma duodenale, 806, 807, 808,
809. *See* Hookworm.
- Anodynes, gastric, 270
- Anorexia, electricity in, 215
nervous, 416
treatment of, 417
- Anoscope, Hirschman's, 830
- Anthrax pustule, 334
- Antidysenteric serum in epidemic dys-
entery, 727
- Antifermentative diet, 181
- Antifermentatives in intestinal diseases,
279
- Antilytic serum in gastric ulcer, 306
- Antiperistalsis of intestine, 63
- Antiputrefactive diet, 174
- Antipyrin, occasional oral effects of, 301
- Antiseptic diet, 174, 689

- Antiseptic mouth washes, 306
value of milk, 174
- Antiseptics in gastric diseases, 272
in intestinal diseases, 279
- Antitryptic reaction in gastric carcinoma, 544
- Anus, artificial, 729
diseases of, 851. *See* Anal, and Pruritus.
fissure of, 858
- Aperient effect of sugars, 284
value of fruits, 284
- Aphthæ, 307
chronic, 308
treatment of, 308
tropical, 308
- Appendicitis, 767
acute, 767
bacteria of, 767
Blaisdell's sign of, 770
Blumberg's sign of, 770
cutaneous hyperesthesia in, 769
diagnosis of, 147, 769
diet in, 777
etiology of, 767
ice in, 776
ingestion of water in, 774
intestinal irrigation in, 774
leukocytosis in, 775
Meltzer's sign of, 770
Murphy drip in, 774, 778
non-surgical treatment of, 776
Ochsner's method of treating, 774
operative mortality of, 773
opium in, 778
from pinworms, 767
point of pain in, 770
purgation in, 776
sigmoidal disease and, 786
symptoms of, 768
time of operation in, 774
treatment of, 773
internal medical, 776, 777
surgical, 773, 777, 778
vaccine, 780
- as a cause of nervous dyspepsia, 419
- cecal tuberculosis and, 741
- cecum mobile and, 770
- chronic, 771
Aaron's sign of, 773
Bastedo's dilatation test for, 773
cecal distention test for, 772
diagnosis of, 772
diet in non-operative cases of, 779
drinking cures in, 779
Morris' pressure point in, 773
movable kidney and, 768
Rovsing's sign of, 772
Rutkevich's sign of, 772
symptoms of, 771
- Appendicitis, chronic, treatment of, 79
destructive, 768
duodenal ulcer and, 705
larvata, 772
periaigmoiditis and, 785, 789
phylacogens in, 780
relation of other diseases to, 767
visceral ptosis and, 768
- Appendicostomy in chronic dysentery, 729
- Appendicular inflammation, 767
- Appendix vermiformis, visualisation of by roentgenography, 187
- Aptyalism, 330
- Aqueous solutions, absorption of, 61
- Argyrosis, 301
- Aromatic products of intestinal putrefaction, 686
- Arsphenamine, 534
in amebic dysentery, 725
- Arteriosclerosis, 528
abdominal angina from, 528
antiscloerin in, 532
diagnosis of, 530
etiology of, 528
gastric hemorrhage from, 528
ulcer from, 528
iodid medication in, 532
pathology of, 529
results of, 528
symptoms of, 529
treatment of, 530
hygienic, 531
medicinal, 531
Trunccek's serum in, 532
- Artificial anus, 729
food preparations, 177, 187
compared with milk, 189
- Ascaris lumbricoides, 800, 801. *See* also Round worm.
- Ascites, hepatic cirrhosis and, 592
paracentesis in, 595
potassium bitartrate in, 595
- Aspiration method of obtaining stomach contents, 68, 206
stomach tube, Chase's, 206
- Asthenia universalis congenita, 558
- Astringent food and drink, 177
- Astringents in hyperchlorhydria, 435
in intestinal diseases, 172, 276
- Atonic constipation. *See* Constipation.
- Atony of esophagus, 386
gastro-intestinal, pilocarpin in, 392
of stomach, 95, 473. *See* also Motor insufficiency.
faradization in, 215
high-frequency current in, 219
shown by roentgenography, 140
- Atrophy of liver, 598
- Atropin as an evacuant, 283
in gastric diseases, 271, 392, 435, 441
in ileus, 754
in vagotonia, 390, 392

- Atropin *vs.* pilocarpin, 271
 Atwater's table of food values, 153-155
 Atsperger's rectal refrigerator, 848
 Auerbach's plexus, 63
 Autolavage, 201
 Autonomic nervous system, 388
 Awltail, 803
- B**
- BACILLI**, Boas-Oppler, in diagnosis, 92
 intestinal, that will not grow on gelatin, 177
 tubercle, demonstrated in feces, 122
- Bacteria** in feces, 118
 in intestine, 122, 686
 in mouth, 269, 700
 pathogenic affinities of, 293
 transmutation of, 291, 684
- Bacterial** excitants of intestinal peristalsis, 62
 fermentation, 699
 growth in the intestine, 686, 699
 preparations from feces, 118
 putrefaction, 700
 vaccines in appendicitis, 780
 in chronic intestinal catarrh, 649
 in enteritis membranacea, 656
 in gastric ulcer, 506
 in pruritus ani, 856
 in ulcerative colitis, 735
- Bacteriology** of bile ducts and gall bladder, 607
 of duodenum, 107
- Ball's** operation in pruritus ani, 857
- Bandage**, abdominal, Aaron's, 575
 adhesive plaster, 578
 Friessnitz, 250
- Barium** or bismuth in roentgenography, 135
- Bastardo's** dilatation test for chronic appendicitis, 773
- Baths** in cirrhosis of liver, 594
 cold entire pack, 248
 half, 248
 indications for, 249
 medicated, 249
 mineral, 256
 mud, 257
 prolonged, 249
 rub-off, 247
 sea, 256
 warm entire pack, 249
 wet rub, 247
- Bead** test for gastro-intestinal motility, 129
- Beans** in diet of gastric patients, 167
- Beef** tea, 161, 194 *See* Bouillon cubes
- Beets** in gastric diseases, 166
- Belladonna** in gastric diseases, 271
 in vagotonia, 392
- Belt**, adhesive-plaster, 578
 sign for abdominal bandage, 575
- Benedict's** effervescence test for gastric acidity, 88
- Benzidin** test for occult blood, 123
- Benzyl benzoate** in amebic dysentery, 724
- Beverages** in constipating diet of intestinal diseases, 172
- Bile**, 58
 acids, 58
 constituents of, 58
 daily secretion of, 58
 duct, agglutination of, 610
 obstruction, indications of, 109
 ducts, dilatation of, 615
 diseases of, 607
 drainage of, 104
 hemorrhage into, 614
 neoplasms of, 614
 diagnosis of, 615
 symptoms of, 615
 treatment of, 615
 parasites of, 616
 in duodenal contents, significance of, 109
 enemata, 226
 function of, 58
 pigments, 58
 in stool, 122, 637
 secretion, HCl and, 259
 in stomach, 66
- Bilharzia**, 817
- Biliary** calculi from trematodes, 818
- Bilirubin** in feces, significance of, 122, 646
- Bismuth** in gastric diseases, 265, 266
 in roentgenography, 137, 138, 139
 salts, 265
 stomatitis, 300
- Bitter** waters, 254
- Bitters**, 266
- Blisters** from hot applications, how to avoid, 250
- Blood** in feces, demonstration of, 123
 occult, in feces, benzidin test for, 123
 phenolphthalein ring test for, 124
 significance of, 123
 spectroscopic detection of, 123
 in intestinal tuberculosis, 133, 738
 in stomach contents, 66, 86
 Weber's guaiac test for, 86
 test in diagnosis of duodenal ulcer, 709
- Blood-iron** preparations, 188
- Blood-sugar** in gastric carcinoma, 545
- Blumberg's** sign of acute appendicitis, 770
- Boas' extra-anal** treatment for hemorrhoids, 827
 rectal electrode, 231

- Boas' stomach electrode, 216
 test breakfast 67
 Boas-Oppler bacillus in gastric carcinoma, 92
 Bothriocephalus latus, 794, 797
 Bougie, cannula and, 371
 Crédé's, 838
 esophageal, 350
 Wales, 733
 Bouillon cubes, value of, 195
 Bread in gastric diseases, 165
 value of different kinds of, 165
 Breakfast, test, 67
 Bromid salivation, 301
 Bromids in gastric diseases, 269
 Buccal fundus, carcinoma of, 344
 phlegmons of, 330
 Buhma, 415
 electricity in, 215
 treatment of, 416
 Burns of esophagus, 357
 of mouth, 304
 Butter as an article of diet, 152
 Buttermilk in gastric disease, 104
 belly, 811
- II
- CALCULI of pancreas, 633
 salivary 327
 Calomel as an intestinal antiseptic, 280
 as a purgative, 285
 Caloric requirements in health, 156
 value of foods, 151
 Cancer. See Carcinoma.
 Cancerous, 550
 Cancerodin, 550
 Cannula indica as a gastric sedative, 269
 Cannula, esophageal, 371
 Capsule method, double, of administering HCl, 260
 Caput medusæ, 592
 Carbohydrate absorption, 61
 digestion in achlorhydria, 85
 in hyperchlorhydria, 85
 in stomach, 85
 fermentation, organisms responsible for, 181
 preparations, 192
 Carbohydrates in chronic pancreatitis, 628
 in food curcs, 572
 hydrochloric acid and, 630
 Carbon dioxide emata, 226
 Carcinoma of bile ducts, 611
 diagnosis of, 615
 symptoms of, 615
 treatment of, 615
 of buccal fundus, 344
 of cardia, 543
 treatment of, 551
 of colon, 764
 diagnosis of 764
 of duodenum, 762
 diagnosis of, 763
 symptoms of, 763
 types of, 763
 of esophagus, 362
 alimentation in, 372
 diagnosis of, 363
 predisposing factors in, 363
 radium in, 364
 roentgenography in, 138
 symptoms of, 363
 treatment of, ameliorative, 373
 by radiation, 373
 surgical, 372
 of gall bladder, 614
 diagnosis of, 615
 symptoms of, 615
 treatment of, 615
 of ileum, 764
 of intestine, 761
 in England and Japan, 761
 heredity of, 762
 incidence of, 761
 location of, 761, 762
 pathology of, 762
 in Prussia, 761
 roentgenography in, 764
 statistics on operative treatment of, 765
 test-diet stool findings in, 134
 treatment of, 765
 in Vienna, 761
 of jejunum, 764
 of lps, 342
 of liver, 600
 of malar mucosa, 344
 of maxilla, 346
 of mouth, 342
 relation of leukoplakia to, 342
 of palate, 344
 of rectum, 832
 complications of, 833
 diagnosis of, 833
 inoperable, 834
 radium in, 834
 symptoms of, 832
 treatment of, 833
 surgical, 833, 834
 symptomatic, 834
 and sarcoma of stomach, differentiation of, 554
 of stomach, 97, 537
 adenoid, 540
 age incidence of, 539
 beginning, 543
 blood-sugar tolerance test for, 545
 Boas-Oppler bacilli in, 543
 cardiac stenosis in, 552
 colloid, 540
 complications of, 541

- Carcinoma of stomach, cytodiagnosis:**
 of, 87
 development of, 537
 diagnosis of, 86, 542
 differential, 554
 gelatinous, 540
 heredity in, 539
 incidence of, 537
 medullary, 540
 occult hemorrhage in, 543
 pathology of, 539
 predisposing factors in, 537
 roentgenography in, 142
 scirrhus, 540
 serologic reactions in, 543
 Abderhalden, 544
 antitryptic, 544
 hemolytic, 543
 miostagmin, 544
 stomach contents in, 543
 symptoms of, 541
 territorial incidence of, 537
 test-diet stool findings in, 131
 tests for, 86. *See also* Serologic reactions, above
 cytodiagnostic, 87
 Gluzinski's, 88
 glycyltryptophan, 87
 Salomon's, 86
 Wolff-Junghans, 87
 treatment of, 546, 551
 dietetic, 547, 551
 internal, 546
 by lavage, 548
 medicinal, 549
 arsenic, 550
 autolysin, 550
 cancorin, 550
 cancerodin, 550
 condurango, 549
 methylene blue, 550
 mineral waters, 549
 physical, 549
 radium in, 550
 Roentgen ray in, 550
 of tongue, 342
 of uvula, 344
Carcinoma in male and female, 761
Cardiac carcinoma, 543
 dilator, Myer's, 397
Cardialgia, 410
Cardiospasm, 393
 diagnosis of, 394
 fluoroscopic, 137
 oil cure for, 397
 prognosis of, 395
 symptoms of, 394
 treatment of, 396
 by dilatation, 397
 electrical, 298
 psychic, 396
 surgical, 398
 with sounds and bougies, 397
Carlsbad waters, 253, 264
 in catarrhal jaundice, 611
Carlsbad waters in cholelithiasis, 620
Carmin test of intestinal motility, 128
Carrigen, 188
Castor oil as a purgative, 286
Catalytic action of enzymes, 53
Cataplasms, 250
Catarrh, alimentary, 635
 gastric and intestinal, furruginous
 waters in, 254. *See also* Intestinal catarrh.
Cathartics by enema, 227
Cauterization in diseases of mouth, 298
Cavernous angiomas of mouth, 336
Cecal distention test for chronic appendicitis, 772
Cecum mobile, 146, 560
 Roentgen-ray diagnosis of, 146
 tuberculosis of, 740
 appendicitis and, 770
 symptoms of, 740
 treatment of, 741
 volvulus of, diagnosed by Roentgen ray, 147
Cells of stomach and their function, 54
Cellulose absorption, 61
 behavior of, in intestine, 183
 bread, 184
 in cauliflower, spinach, and cabbage, 183
 in diet, 179
 digestibility of, 183
 digestion of, 61, 183
 ferment, 678
 hemicellulose and, 61, 184
 as a laxative, 184
Centenarians in Bulgaria and Germany, 165
Cervical laceration, nervous dyspepsia and, 419
Charcoal test of intestinal motility, 128
Chase's aspiration tube, 206
Cheeks, affections of, 333, 335
Cheese in gastric diseases, 165
Cheilitis, acute, 334
 chronic, 334
 exfoliativa, 334
 glandularis, 334
Chemical examination of feces, 116
 of stomach contents, 74
Childbirth, gastroenteroptosis and, 557, 581
Chloral hydrate as a gastric sedative, 269
Chloroform as a gastric anodyne, 270
 water irrigations, 227
Chlorophyll test of gastric motility, 91
Chocolate, digestibility of, 169
 "Kraft," 194
Cholangitis, 607
 cholecystitis and, diagnosis of, 610
 etiology of, 607
 hemocones in, 611
 pathology of, 607
 sequelæ of, 609
 suppurative, etiology of, 613

- Cholangitis, suppurative, pathology of,** 613
 symptoms of, 614
 treatment of, 614
 symptoms of, 608
 treatment of, 611
 antipruritic, 613
 Carlsbad, 611
 diet, 613
 glycerin, 612
 magnesium sulphate, 612
- Cholecystitis, 608. See also Cholangitis.**
 adhesions from, 611
 anatomic effects of, 608
 diagnosis of, 610, 619
 duodenal contents in, 109
 ulcer and, 708
 nervous dyspepsia and, 419
 pathogenesis of, 607
 symptoms of, 608
 treatment of, 611
- Cholelithiasis, 616**
 calomel in, 552
 Carlsbad cure for, 620
 cholagogues and antiseptics in, 621
 diagnosis of, 618
 drainage, non-surgical, in, 621
 duodenal ulcer and, 708
 etiology of, 617
 heredity in, 617
 HCl secretion in, 85
 nervous dyspepsia and, 419
 roentgenography in, 150, 618
 symptoms of, 618
 treatment of, 619
 dietetic, 620
 medicinal, 621
 mineral water, 620
 by non-surgical drainage, 621
 surgical, 622
- Cholemia, 610**
- Cholera morbus, 638, 642. See also**
 Intestinal catarrh.
 hypodermoclysis in, 643
 pain and vomiting in, 643
 sea-water in treatment of, 642
 nostras, 638. *See* **Intestinal**
 catarrh, acute.
- Cholesterol, 59**
- Cholesterolemia, 617**
- Chologen in cholelithiasis, 621**
- Chondroma of maxillæ, 345**
 of mouth, 336
- Chondrosarcoma, maxillary, 346**
- Chyme, rate of movement of, 64**
- Cirrhosis of liver, atrophic, 590**
 etiology of, 590
 levulose test in, 593
 pathology of, 590
 phthalein test in, 593
 prognosis of, 593
 symptoms of, 591
 tests for lipase in, 592
 treatment of, 594
 medicinal, 594
- Cirrhosis of liver, atrophic, treatment**
 of, surgical, 595
 urobilin test in, 593
 in course of other diseases, 598
 from trematodes, 818
 hypertrophic, 596
 in bronze diabetes, 597
 diagnosis of, 597
 etiology of, 596
 prognosis of, 597
 symptoms of, 596
 treatment of, 597
 biliary, 598
- Climatic cures, 256**
- Coagulen, 715**
- Coagulose, 516**
- Cocain hydrochlorid as a gastric seda-**
 tive, 269
- Coccygodynia, 850**
 treatment of, 850
 cauterization, 850
 massage, 850
 sedative, 850
- Cocoa, acorn, 194**
 nutritive value of, 169
 protein-milk-salt, 193
- Cod-liver oil, 194**
- Coffee, effect of, on digestion, 169**
- Coils, Leiter's, 251**
- Cold entire pack, 247**
- Colectomy in various diseases, 697**
- Colica flatulenta, 783**
- Coliques salivaires, 327**
- Colitis, 635. See also Enteritis and**
 Intestinal catarrh.
 acute, 635
 chronica ulcerosa, 730
 dry treatment of, 237
 membranous, high-frequency cur-
 rent in, 219
 mucous, 652
 enteroptosis and, 653
 spastic constipation and, 670
 vagotonia and, 653
 ulcerative, 730
 bacteria in etiology of, 730
 complications of, 731
 creosote and cod-liver oil in,
 732
 diagnosis of, 731
 lavage and irrigation in, 733
 opium and pantopon in, 732
 pathology of, 730
 perisigmoiditis and, 731
 symptoms of, 731
 treatment of, 732
 "dry," 734
 local, 733
 Matthews', 733
 vaccine, 735
- Colon, carcinoma of, 764. See Carci-**
 noma of colon.
 deviations from normal position
 of, 146
 dilatation of, 790

- Colon, dilatation of, in boys, 791
 etiology of, 791
 idiopathic, 790
 prognosis of, 792
 roentgenography in diagnosis of, 791
 symptoms of, 792
 treatment of, 793
 inflammation of, 635. *See* Colitis.
 irrigation of, 221
 normal motility of, 145
 position of, 145
 roentgenography of, 145
 transverse, displacement of, 146
 tube, 220
- Colonic irrigation, posture in, 221
 technic of, 221
 stasis, roentgenography in, 147
- Coloptosis, 559, 560
 roentgenographic diagnosis of, 146
- Compresses, 250
- Condurango bark as a stomachic, 267
- Connective-tissue adhesions, massage in, 208
 digestion, 674
 in feces, significance of, 119, 674
- Constipating diet, 172
- Constipation, acute, water enemata in, 227
 agar in treatment of, 184, 662
 atonic, 659
 etiology of, 659
 magnesium sulphate subcutaneously in, 666
 mineral waters in, 667
 roentgenography in, 661
 symptoms of, 660
 test-diet stool findings in, 133, 661
 treatment of, 661
 dietetic, 182, 184, 185
 by enemata, 223, 668
 electrical, 219
 general, 665
 hormone, 667
 lavage, 666
 liquid petrolatum, 664
 massage, 208, 664
 medicinal, 666
 surgical, 668
 with agar, 662
 with grapes, 664
- chronic, 659
 atonic, agar in, 185, 662
 diet in, 185
 oil enemata in, 223
 paraffin enemata in, 225
 bile enemata in, 226
 carbon dioxid enemata in, 226
 cathartics by enema in, 227
 electricity in, 230
 enemata in, 222
 etiology of, 659
 feces in, 660
 massage in, 214
- Constipation, chronic, oil enemata in, 223
 paraffin enemata in, 225
 rectal friction in, 229
 massage in, 229
 tampons in, 230
 Swedish manipulation in, 228
 symptoms of, 660
 test-diet stool of, 661
 transverse colon and, 146
- cleansing enemata in, 220
 diet in, 182-186
 early, from megacolon, 791
 fragmentary, 672
 treatment of, 672
 fruit in diet of, 184
 glycerin enemata in, 222
 hemicellulose in, 184
 intestinal irrigation in, 666
 lavage, duodenal, in, 107
 liquid petrolatum in, 228
 massage in, 214
 mineral waters in, 254
 movement of bismuth mass in, 146
 nervous dyspepsia and, 419
 perisigmoiditis and, 785
 rectal, retention of bismuth in, 146
 spastic, 668
 action of opium in, 672
 cause of, 668
 colitis and, 670
 diagnosis of, 670
 innervation of intestine in, 668
 irritating foods contra-indicated in, 186
 purgatives contra-indicated in, 672
 roentgenography in, 669
 sex incidence of, 669
 symptoms of, 669
 test-diet stool findings in, 134, 670
 treatment of, 670
 antispastic, 672
 dietetic, 670
 by dilatation of anus, 671
 electric, 219, 671
 fruit sugars in, 185
 by oil enemata, 223, 671
 with hot and cold air
 douche, 671
 vegetative nervous system and, 671
- Constitutional diseases, effect of, upon mouth, 301, 302
- Contrary innervation, law of, 65
- Convulsions from overeating, lavage in, 198
- Corpus luteum in pernicious vomiting of pregnancy, 405
- Corrosions of esophagus, 357
- Corsets, abdominal, 580
- Crawcour's esophageal sound, 367
- Cream protein mixture, Pfund's, 194
- Crédé's bougie, 838

- Cr  de's method of gastric massage, 210
 Crises, gastro-intestinal, 689
 Croton oil in atonic constipation, 667
 Crystals in stomach contents, 94
 Curve of gastric digestion, 79
 Cynorexia, 415
 Cystitis from trematodes, 818
 Cysts of bile ducts, 616
 dermoid, of mouth, 337
 of esophagus, 362
 from glands of mouth, 338
 maxillary, 345
 of pancreas, 632
 at root of tongue, 339
 of stomach, 555
 Cytase, 59, 61
 Cytodiagnosis of gastric carcinoma, 87
- D**
- DEFENSE** musculaire in appendicitis, 769, 775
 in duodenal ulcer, 709
 Deglutition sounds, 350
 Delineator string in diagnosis of cardio-spasm, 396
 of pylorospasm, 399
 Dentifrices recommended, 294, 295
 Dermoid cysts in esophagus, 362
 in mouth, 337
 Dextrose, 50
 Diabetes, bronze, with hypertrophic cirrhosis of liver, 597
 mellitus, lavage in, 198
 pyorrhea and, 303
 Diagnosis and pathologic stools, 119
 Diaphragm, displacement of, 560
 Diarrhea, acute, 635
 chronic, 673
 agar-tannin in, 278
 condition of stomach and, 674
 diet in, 172
 ferruginous waters in, 256
 from decomposable fluids, 173
 from transudation of serum, 173
 gambur in, 278
 gastrogenic, 173, 180, 673
 bacterial processes in, 675
 etiology of, 674
 examination of stomach contents in, 676
 fecal findings in, 673
 lavage in, 677
 symptoms of, 675
 treatment of, 676
 dietetic, 676
 medicinal, 677
 with acids or alkalis, 676
 with reference to stomach, 676
 in intestinal tuberculosis, 738
 hematoxylin in, 278
 milk somatose in, 277
 nervous, 680
 Diarrhea, nervous, diagnosis of, 681
 etiology of, 680
 psychogenic, 680
 reflex, 680
 symptoms of, 681
 test-diet stool findings in, 134, 681
 treatment of, 681
 dietetic, 682
 mental, 681
 with epinephrin, 682
 remedial value of, in enteritis, 638
 spastic, 670
 strychnin in, 407
 tannin preparations in, 277
 tubular, 652
 Diastase in feces, Wohlgemuth's test for, 127
 Diastatic ferments, medicinal use of, 263
 Diet in acute appendicitis, 777
 dysentery, 722
 enterocolitis, 639
 gastritis, 452
 infectious gastritis, 454
 intestinal catarrh, 639
 in anal fissure, 860
 antidiabetic, in pancreatic fistula, 630
 antifermentative, 181
 antiputrefactive, 174
 antiseptic, 174, 689
 astringent, 177
 in atonic constipation, 183, 184, 661
 in cardiospasm, 393
 cellulose in, 179, 183
 in cholangitis and cholecystitis, 613
 in cholelithiasis, 620
 in chronic appendicitis, non-operative, 779
 dysentery, 727
 gastritis, 466
 gastrorrhea, 444
 intestinal catarrh with constipation, 650
 with diarrhea, 647
 pancreatitis, 627
 constipating, 172
 in constipation, 182-186
 spastic, 670
 in diarrhea, 172, 639
 gastrogenic, 676
 nervous, 682
 in dilatation of colon, 793
 in duodenal ulcer, 711
 in enteritis membranacea, 654
 in flatulence, 701
 in gastric atony, 475
 carcinoma, 547, 551
 diseases, 151
 ulcer, 499, 500
 in gastritis, acute, 452
 infectious, 454
 in gastroenteroptosis, 569, 571

- Diet in hemorrhoids, 823
 in hyperchlorhydria, 432
 in hypersecretion, 444
 in ileus, 749
 instruction of patient regarding, 169
 in intestinal catarrh, 639, 647, 650
 diseases, 172
 fermentative dyspepsia, 181, 177
 hemorrhage, 172
 putrefaction, 174, 178
 stricture, 759
 toxemia, 689
 tuberculosis, 739
 ulcer, 172, 711, 714, 732
 laxative, 182
 milk as an article of, 174
 in motor insufficiency of first degree, 475
 of second degree, 481
 in mucous colitis, 654
 in nervous dyspepsia, 421
 in pancreatic affections, 627, 629
 and patient's preference, 152
 in pruritus ani, 853
 in putrefactive intestinal diseases, 174
 in rectal paralysis, 849
 prolapse, 846
 Schmidt, 112, 179, 180
 sustaining, 570
 in thread worm, 805
 in typhoid fever, 713
 in ulcerative colitis, 732
 vitamin in the, 157
 Dietary instructions, 169
 regulations and lists, 152
 Dietetic treatment and stomach tube, 151
 Digestibility of foods, 156
 Digestion, activators of, 53, 157
 carbohydrate, in stomach, 85
 effect of alcohol on, 168
 gastric, 52
 gelatin test of, 84
 intestinal, 56, 61
 kinases in, 53
 Pawlow's experiments in, 52, 54, 56
 physiology of, 49
 salivary, 49
 shown by food beads, 129
 Dilatation of anus in rectal stricture, 839
 in spastic constipation, 672
 of biliary organs, 615
 of colon, idiopathic, 790
 roentgenography in, 148
 of esophageal stricture, 368
 of esophagus, 380
 of rectal stricture, 839, 841
 of stomach, 95, 478
 acute, 486
 shown by roentgenography, 140
 Dilatation of stomach, stomach contents in, 95
 treatment of, 486
 by lavage, 487
 by massage, 208
 Dilators, esophageal, 370
 rectal, 839, 840
 Dimethylamidoazobenzol test for free hydrochloric acid, 75
 Dinner, test, 68
 Diphtheria in esophagus, 356
 Distoma hematobium, 816, 817
 hepaticum, 817
 lanceolatum, 817
 Diverticula of esophagus, 365, 374
 of sigmoid, 786, 787, 788
 Diverticular sounds, 376
 Diverticulitis, roentgenography of, 148
 sigmoidal, 786
 etiology of, 788
 pathology of, 788
 Dochmius duodenalis, 806
 Dorsal pain of gastric ulcer, 491
 Double capsule method of administering hydrochloric acid, 260
 Douches, 251
 intestinal, 228
 Draining gall bladder and bile ducts, 104
 Drinking cures, 254, 256
 Dropsy, Karell cure for, 163
 Drum-belly, 403
 Dry treatment of colitis, 237
 of intestinal catarrh, 237
 of ulcerative enteritis, 734
 Dumb-bell pessary for hemorrhoids, 826
 Duodenal alimentation, Einhorn's tube for, 500
 in gastric ulcer, 500, 502, 508
 Morgan's modification of Einhorn's, 502
 technic of, 501
 bag, Hemmeter's, 98
 bucket, Einhorn's, 91
 cap, 143
 carcinoma, 762
 contents after test meal, 101
 characteristics of, 101
 examination of, 98
 in duodenal ulcer, 110
 in duodenitis, 110
 in liver and gall bladder lesions, 101
 pancreatic juice in, 109
 in pancreatitis, 109
 in pernicious anemia, 110
 results of direct examination of, 109
 significance of bile in, 109
 of typhoid carriers, 110
 in typhoid fever, 110
 urobilin and urobilinogen in, 101
 enzymes, determination of, 103
 examination for, 102

- Duodenal enzymes in pathologic conditions**, 103
feeding, 500. *See* Duodenal alimentation.
fluid, bacteria in, 107
lavage, 105
 indications for, 107
 for intestinal stasis, 107
 irrigating fluid employed in, 106
 Jutte's apparatus for, 104, 105
 technic of, 106
medication, direct, 104
tube, an aid to the passage of, 100
 determination of, in duodenum, 100
 Einhorn's, 98, 500
 Gross's, 100
 Jutte's, 100
 Kuhn's, 98
 Palefski's, 100
 ulcer, 705. *See* Ulcer, duodenal.
Duodenitis, duodenal contents in, 110
Duodenum, bacteriology of, 107
 displacement of, 559
 Hemmeter's apparatus for entering, 98
 peptic ulcer of, 705
See also Duodenal.
Dyschezia, 146, 666
Dysenteric pains, epinephrin in, 725, 726, 727
Dysentery, acute, 719
 catechu bark in, 725
 complications of, 721
 epinephrin irrigations in, 726
 iodoform in, 726
 magnesium sulphate in, 725
 pathology of, 720
 prognosis of, 721
 prophylaxis of, 721
 silvol enemata in, 726
 symptoms of, 720
 tannic acid enteroclysis in, 726
 treatment of, 722
 dietetic, 722
 medicinal, 722
 amebic, arsphenamine in, 725
 benzyl benzoate in, 724
 bismuth subnitrate in, 726
 emetin in, 723
 emetin-bismuth iodid in, 724
 epinephrin in, 725
 from trematodes, 818
 ipecac in, 722
 simaruba bark in, 725
 uzara in, 725
 bacillary, 727
 chronic, 719, 727
 appendicostomy in, 729
 castor oil in, 728
 diet in, 727
 emetin or ipecac in, 728
 iodoform enemata in, 726
 lactic acid cultures in, 727
Dysentery, chronic, olive oil in, 728
 pathology of, 720
 site of lesions in, 728
 treatment of, 727
 medicinal, 728
 surgical, 728
 ulcers in, 720
 complications of, 721
 differentiation of acute and chronic, 720
 endemic, 719
 epidemic, 719
 antidysenteric serum in, 727
 from parasites, 719
 from trematodes, 818
 microbic causes of, 719
 pathology of, 720
 test-diet stool findings in, 133
Dyspepsia, intestinal fermentative, 181, 677
 etiology of, 678
 oxygen insufflation in, 679
 test-diet stools in, 134, 678
 treatment of, 679
 nervous, 418
 causes of, 418, 419
 definition of, 418
 eye strain and, 418
 HCl secretion in, 420
 lactovegetable diet list for, 422
 massage in, 208, 423
 obstetric lacerations and, 419
 prognosis of, 420
 prophylaxis of, 420
 sea-water therapy in, 423
 apparatus for application of, 424
 symptoms of, 419
 treatment of, 421
 dietetic, 421
 etiologic, 421
 medicinal, 425
 physical, 423
 sea-water, 423
 umbilical, 428
Dyspeptine, 262
- E**
- ECHINOCOCCI in bile ducts**, 616
 in liver, 602, 604
 diagnosis of, 603
 etiology of, 602
 symptoms of, 603
 treatment of, 604
Echinococcus multilocularis in liver, 604
Eclampsia, lavage in, 198
Eczema, labial, 317
 oral carcinoma and, 342
Effervescence test of gastric function, 88
Effleurage, 213
Egg protein preparations, 192
Eggs in diet, 161
 food value of, 162

- Eggs, preparation of, 161
 Einhorn's agar tubes, 102
 apparatus for stomach douche, 205
 bead test of gastro-intestinal motility, 129
 duodenal alimentation, 500
 bucket, 91
 tube, 98, 500
 intra-gastric electrode, 217
 pyloric dilator, 400, 485
 stomach bucket, 70
 test meal before examining duodenal contents, 101
 Elbrecht's heating apparatus for proctoclysis, 240, 241, 242, 243
 Electricity in acute intestinal catarrh, 651
 in digestive disorders, 219
 in gastralgia, 215, 219
 in gastric disorders, 214, 215, 219
 in gastroenteroptosis, 574
 in gastropnoxis, 215
 in intestinal diseases, 230
 in mucous colitis, 656
 in vomiting, hysterical, 215
 of pregnancy, 215
 Electrization in chronic constipation, 230
 extraventricular, 219
 technic of, 219
 intraventricular, 216
 apparatus for, 216
 of stomach, indications for, 215
 Electro-colloids for intravenous injection, 581
 Electrode and stomach tube combined, Stockton's, 218
 Boas', 216, 231
 Einhorn's, 217
 hemorrhoidal, Williams', 832
 intra-gastric, 216, 217
 rectal, 230, 231, 232
 Wegele's, 217
 Zweig's, 231
 Electrolysis in treatment of internal hemorrhoids, 831
 Electrotherapy in cardiospasm, 398
 Embolic ulcers of intestine, 741
 Embryonic membrane, 561
 Emetin in amebic dysentery, 723
 Emetin-bismuth iodid in amebic dysentery, 724
 Emollients in gastric diseases, 272
 Empyema of gall bladder, 616
 Endothelioma of mouth, 340
 Enemata in atonic constipation, 220, 666
 bile, 226
 carbon dioxide, 226
 apparatus for administering, 227
 of cathartics, 227
 cleansing, apparatus for administering, 221
 technic of, 220
 Enemata, glycerin, 222
 in intestinal stenosis, 227
 nutrient, 243
 oil, 223
 apparatus for administering, 224, 225
 paraffin, 225
 Enemator, Roberts' oil, 225
 Zweig's oil, 223
 Enteralgia, causes of, 783
 nervosa, 783
 treatment of, 784
 Enteritis, chronic, 644. *See also* Intestinal catarrh, chronic.
 membranacea, 652
 appendicitis and, 587
 etiology of, 652
 pathology of, 652
 surgery in, 657
 symptoms of, 653
 test-diet stool findings in, 133
 treatment of, 653
 anodyne, 658
 antispasmodic, 656, 658
 dietetic, 654
 electric, 656
 hydrotherapeutic, 656
 irrigation, 655
 laxative, 656, 658
 massage, 656
 sea-water, 657
 vaccine, 656
 with anemia, 654
 mucomembranous, 652
 ulcerative, 730
 bismuth subgallate in, 734
 complications of, 731
 course of, 731
 creosote and cod-liver oil in, 732
 diagnosis of, 731
 diet in, 732
 "dry treatment" of, 734
 etiology of, 730
 exudates from, 731. *See* Perisigmoiditis.
 irrigations in, 733
 lavage in, 733
 opium in, 732
 pathologic anatomy of, 730
 prognosis of, 731
 treatment of, 732
 Matthews', 733
 surgical, 735
 vaccine, 735
 Enterocolitis, acute, 635. *See* Intestinal catarrh.
 chronic, 644
 Enterokinase, 59
 Enteroptosis, 557
 mucous colitis and, 653
 nervous dyspepsia and, 419
 Enterospasm, feces in, 781
 symptoms of, 781
 treatment of, 781

- Enzymes, 53**
 catalytic action of, 53
 duodenal, estimation of, 102
 examination for, 83
 in stomach, tests for, 83
- Eosinophilia in hookworm disease, 811**
 in round worm, 800
 in trichinosis, 819
- Epigastric hernia and nervous dyspepsia, 419**
- Epinephrin (adrenalin) in anal fissure, 861**
 in diagnosis of pancreatic insufficiency, 626
 in dysenteric pains, 725, 726, 727
 in esophageal corrosions, 358
 in gastric hemorrhage, 271, 517
 in hemorrhoids, 823
 irrigations in dysentery, 726
 in nervous diarrhea, 682
 in pylorospasm, 400
 in vomiting of pregnancy, 404
 vs. pilocarpin, 390
- Epulis, 346**
- Erepsin, 59**
- Erosions of mouth, 303**
 of stomach, 97, 521
 acute or hemorrhagic, 521
 chronic, 521
 diagnosis of, 522
 effect of, on stomach contents, 97
 etiology of, 521
 pathology of, 522
 prognosis of, 523
 symptoms of, 522
 treatment of, 523
 hydrotherapeutic, 523
 local, 523
 with bismuth, 524
 with silver nitrate, 523
 with suprarenal gland, 524
- Eructation, nervous, 401**
- Erysipelas, scleroma and, 315**
- Erysipelatous stomatitis, 307**
 treatment of, 307
- Erythema exudativum multiforme of oral mucosa, 318**
- Erythrodextrin, 50**
- Escalin in gastric hemorrhage, 518**
- Esmarch's rectal truss, 847**
- Esophageal anesthesia, 385**
 atony, 386
 bougie, 350
 and cannula (Leyden and Renvers), 371
 carcinoma, 362. *See* Carcinoma of esophagus.
 dilatation, congenital, 382
 diagnosis of, 381
 etiology of, 380
 prognosis of, 381
 roentgenography in, 381
- Esophageal dilatation, symptoms of, 380**
 treatment of, 381
- dilator, Schreiber's, 370**
 Senator's, 370
 Sippy's, 369, 370
- diphtheria, 356**
- diverticula, 365, 374**
 pulsion, 374
 diagnosis of, 375
 roentgenographic, 138
 prognosis of, 375
 symptoms of, 375
 treatment of, 376
 sound treatment of, 365
 traction, 374
- hemorrhage, 383**
- hyperesthesia, 384**
- malacia, 383**
- neuroses, 384**
- paralysis, 385**
- perforation, 383**
- rupture, 383**
- sound (Crawcour's), 367**
- sounds, 365, 376**
 introduction of, 365, 367
- spasm, 378**
 roentgenologic appearance of, 137
 symptoms of, 378
 treatment of, 379
- stricture, 364**
 alimentation in, 372
 congenital, 383
 dilatation of, 368
 from dilatation. *See* Esophageal dilatation.
 from diverticula, 374
 from external compression, 378
 from foreign bodies, 377
 from neoplasms, 362
 from spasm of esophagus, 378
 from thrush, 378
 treatment of, general, 373
 sound, 364
 surgical, 372
- syringe, Rosenheim's, 354**
- tuberculosis, 359**
- variola, 357. *See also* Esophagus.**
- Esophagitis, acute, 353**
 treatment of, 353
- chronic, 353**
 treatment of, 354
- exfoliative, 353**
 treatment of, 355
- fibrinous, 355**
 treatment of, 355
- phlegmonous, 356**
 treatment of, 356
- Esophagoscope, 351, 352**
 introduction of, 351
 in locating diverticula, 375
- Esophagus, actinomycosis of, 361**
 anatomy of, 349
 atony of, 386

- Esophagus**, burns of, 357
 carcinoma of, 362. *See* Carcinoma of esophagus.
 cicatricial stricture of, 364
 corrosions of, 357
 cysts of, 362
 dermoids of, 362
 dilatation of, 380
 diseases of, 349
 fibroma of, 362
 foreign bodies in, 377
 gangrene of, 358
 herpes zoster in, 357
 hypertrophy of, 362
 infectious diseases of, 356
 inflammation of, 353
 instrumental examination of, 350
 lipoma of, 362
 myoma of, 362
 neuroses of, 384
 papilloma of, 362
 parasites in, 361
 pemphigus in, 357
 Roentgen-ray examination of, 136
 sarcoma of, 364
 stricture of, 361, 364, 378, 383
 cicatricial, treatment of, 364
 syphilis of, 359
 thrush of, 361
 tuberculosis of, 359
 ulcers of, 358
 peptic, 360
See also Esophageal.
Etat mamelonné, 461
Eucasin, 192
Eumydrin in gastric diseases, 271, 446
 in hypersecretion, 271
Examination of duodenal contents, 98
 for enzymes, 83, 102
 of feces, 111. *See* Feces.
 macroscopic, 115
 microscopic, 115
 of intestine, 143
 of stomach contents, 66
 chemical, 74
 macroscopic, 68
 microscopic, 92
 roentgenologic, 135
See also Stomach contents.
Exanthems, medicinal, 301
Exercise after eating, 170
Experimental ulcer, 706
Expression method of obtaining stomach contents, 68
Eye-strain as a cause of gastric neuroses, 418
 headache and, 418
- F**
- FACIAL** nerve, paralysis of, 320
Faradization of stomach, 215, 218, 219
Fat absorption, 60
 in diet of gastric patients, 152, 162
Fat digestion in chronic pancreatitis, 625
 in feces, 120, 127
 in food cures, 572
 importance of, in the dietary, 572
 limitation of, in chronic pancreatitis, 627
Fat-digestion tests, 127
Fat-splitting ferment, lipase, 56
Fats permissible in stomach diseases, 111
 preparation for digestion of, 56
 retention of, in stomach, 51
Fatty liver, 604
Fauces, effects of rubecula on, 302
Febrilis icterus, 587
 etiology of, 588
 serum treatment of, 588
Fecal analysis, apparatus for making, 114
Feces, carbohydrates in, 121
 composition of, 65
 connective tissue in, 119
 demonstration of ferments in, 125
 diastase in, test for, 127
 in enterospasm, 781
 examination of, 111
 for bacteria, 118
 for blood, 123, 124
 chemical, 116
 for bilirubin, 122
 for dissolved protein, 117
 reaction test, 116
 Schmidt's incubator test, 116
 sublimite test, 116
 Strasburger's fermentation tubes, 117
 in duodenal ulcer, 133, 709
 in gastrogenic diarrhea, 673, 674
 in intestinal fermentative dyspepsia, 677
 for protein, 117
 for pus, 122
 test diet in, 112
 fat in, 120, 127
 fermentation of putrefaction of, 172
 in intestinal catarrh, 637
 mucus in, 121
 muscle remnants in, 120
 normal after test diet, 119
 nuclei in, 126
 potato remnants in, 121
 protein in, 121
 starch in, 121
 steapsin in, 125
 trypsin in, 125
See also Test-diet stool findings.
Feeding in diseases of mouth, 298
Fermentation tubes, Strasburger's, 116
Fermentative dyspepsia, 181
Ferments in feces, demonstration of, 125
Ferruginous waters, 254
Fersan, 188

- Fetor ex ore* from bromids and iodids, 301
 in catarrhal stomatitis, 305
 from esophageal dilatations, 380
 in esophageal diverticula, 375
 in gangrenous stomatitis, 306
 from mercury, 300
 in noma, 307
 to remove, 296
 in pemphigus of oral mucosa, 318
 treatment of, 296
 Fibrolysin in pyloric stenosis, 484
 Fibroma of esophagus, 362
 of maxilla, 345
 of mouth, 335
 of stomach, 555
 Fibromyoma of stomach, 555
 Filtering gastric contents, reason for, 78
 Fissure of anus, 858
 Fistula in ano, 857
 of lower lip, 333
 Flatulence, 698
 aerophagy and, 701
 endogenous gas in, 699
 exogenous gas in, 699
 from cardiac disease, 704
 from food decomposition, 700
 from impeded expulsion of gases, 700
 from intestinal stricture, 704
 from microorganisms, 699
 origin of, 698
 nervous, 700
 pseudo, 700
 relieved by attention and sleep, 701
 treatment of, 700
 dietetic, 701
 by intubation, 703
 by massage, 702
 medicinal, 702
 Flemer's oil enemata in spastic constipation, 223, 671
 Flies in intestine, 820
 Floating kidney, 566
 chronic appendicitis and, 768
 Flour, aleuronat, 191
 dextrinated, 193
 preparations, 193
 Flours in diet, 193
 Fluctuating rib and gastroenteroptosis, 224
 Fluke worms, 816
 Focal infection, 290
 Follicular tonsillitis, 347
 Food, antiseptic, 174
 astringent, 177
 caloric value of, 151
 chemical composition of, 151
 cures, carbohydrates in, 572
 fat in, 572
 proteins in, 571
 preparations, artificial, 177, 187
 cows' milk and, 189
 Food preparations containing fat, 194
 cod-liver oil, 194
 lipanin, 194
 Mering's "Kraft"
 chocolate, 194
 nutrole, 194
 oil of sesame, 194
 Russell's emulsion, 194
 sevetol, 194
 from animal protein, 187
 from carbohydrates, 192
 from egg protein, 192
 from milk protein, 194
 from vegetable protein, 191
 mixed, 193
 acorn cocoa, 194
 hygiamia, 193
 odda, 193
 protein-milk-salt cocoa, 193
 racahout, 194
 products, average composition of, 153, 155
 remnants, pathologic, 119
 requirements in health, 156
 stimulating, 194
 values, 151-169
 Atwater's table of, 153-155
 Foods, digestibility of, 156
 heat value of, 151
 proprietary, 191
 Foot-and-mouth disease in man, 302
 Foreign bodies in esophagus, 377
 Fractional analysis of stomach contents, 78
 Fragmentary constipation, 672
 Friedlieb's stomach tube, 202, 203
 Friedrich's test for HCl in stomach, 89
 Fruit in diet of constipation, 184, 185
 of gastric patients, 167
 Funnel pessary for hemorrhoids, 826
- G**
- GALACTOGEN, 192
 Gall bladder, drainage of, 104
 empyema of, 616
 hydrops of, 616
 neoplasms of, 614
 roentgenography of, 149
 Gall-bladder disease, duodenal bile in, 109
 diseases, 607
 Gallstone colic, treatment of, 619
 ileus, chloroform-water irrigations in, 227
 Gallstones, 616. See also Cholelithiasis, as a cause of nervous dyspepsia, 419
 diagnosis of, 618
 roentgenography in, 150, 618
 non-surgical drainage for, 104, 621
 origin of, 617

- Gallstones, surgical removal of, 622
 symptoms of, 618
 treatment of, 619
- Galvanofaradization of stomach, 218
- Gambir, 278
- Gangrene of esophagus, 358
- Gangrenous stomatitis, 305
 treatment of, 306
- Gas-producing organisms in duodenal fluid, 108
- Gases, fermentative and putrefactive, 700
 in flatulence, endogenous, 699
 exogenous, 699
 from microorganisms, 699
 in intestine, origin of, 699
- Gasterin, 262
- Gastralgia, 266, 270, 410
 anesthesin in, 270
 belladonna in, 271
 bismuth in, 266
 causes of, 410
 chloroform in, 270
 electricity in, 215, 219
 Franke's operation in, 412
 nervous, massage and medicated lavage in, 210
 orthoform-new in, 270
 rhizotomy in, 412
 strychnin in, 266
 treatment of, 410
- Gastralgokenosis, 414
- Gastric analysis, indirect methods of, 88. *See* Stomach contents.
- anodynes, 270
- carcinoma, 97, 537. *See* Carcinoma of stomach.
- catarrh, diet in, 151
 mineral waters in, 255, 256
- crises in intestinal toxemia, 689
- vagus and sympathetic, 412
- digestion, 52
 curve of, 79
 Günzburg's test of, 89
 Sahl's desmoid test of, 90
- dilatation with pyloric stenosis,
 lavage in, 197
 massage in, 208
 postoperative lavage in, 197
- diseases, acidul in, 260
 alcohol in, 168, 267
 alkalis in, 263
 antiseptics in, 272
 hydrogen peroxid, 272
 iodin, 272
 magnesium peroxid, 273
 phenol, 272
 resorcinol, 272
 salicylates, 272
- bismuth in, 263, 266
- bitters in, 266
- diet in, 151
- drugs used incidentally in, 271
- Gastric diseases, drugs used incidentally
 in, atropin, pilocarpin and nicotin, 271
 epinephrin, 271
 eumydrin, 271
- gasterin in, 262
- gastric anodynes in, 270
 sedatives in, 268
 amyl nitrite, 268
 bromids, 269
 cannabis, 269
 chloral hydrate, 269
 cocaine hydrochlorid, 269
 dilute hydrocyanic acid, 269
 nitroglycerin, 269
 hydrochloric acid in, 258
 olive oil in, 273
 orexin in, 267
 pancreatin in, 262
 pepsin in, 258
 silver nitrate in, 267
 strychnin in, 266
 tobacco in, 169
 water in diet of, 168
- glands, tubular, 54
- hemorrhage, 511. *See* Hemorrhage, gastric.
- hyperacidity, 94. *See* Hyperacidity and Hyperchlorhydria.
- hyperesthesia, 413
 silver nitrate in, 413
 Stockton's sedatives in, 414
- irritation, mud baths in, 257
- juice, acidity of, 54
 after ingestion of bread, 53
 of milk, 53
 color of, 73
 consistency of, 74
 determination of, 73
 normal, 54
 odor of, 74
- lavage. *See* Lavage of stomach.
- motility, chlorophyl test of, 91.
See Motor function of stomach and Motor insufficiency.
- mucus, lavage for removal of, 197, 205
- neuroses, 94
 eye-stram and, 418
 mud baths in, 257
- peristalsis and duodenal ulcer, 144
- retention, massage in, 208
- secretion, changes in, due to pathologic conditions, 94
 in duodenal ulcer, 709
 duration of, 55
 meat and, 53
 in nervous dyspepsia, 94
 psychic imitation of, 55
 Sahl's desmoid test for, 90
 stimulants of, 267
 vagus nerve and, 54

- Gastric sedatives, 268
 subacidity, test-diet stool findings in, 131
 tetany, 485
 lavage in, 198
 tonus, roentgenography of, 140
 ulcer, 97. *See* Ulcer, gastric.
- Gastritis, acid, 462
 acute, 95, 449
 infectious, 453
 etiology of, 453
 pathology of, 453
 symptoms of, 454
 treatment of, 454
 dietetic, 454
 by lavage, 454
 medicinal, 454
 simple, 449
 course of, 450
 etiology of, 449
 pathology of, 449
 prophylaxis of, 450
 symptoms of, 450
 treatment of, 450
 dietetic, 452
 by lavage, 198, 450
 medicinal, 452
 sodium chlorid waters in, 253
 stomach contents in, 95
 anacid, 463. *See* Achylia gastrica.
 chronic, 95, 460
 diagnosis of, 462
 etiology of, 460
 HCl secretion in, 95
 massage in, 472
 pathology of, 461
 prognosis of, 463
 stomach contents in, 95
 symptoms of, 461
 treatment of, dietary, 466
 by lavage, 471
 medicinal, 469
 physical, 472
 with mineral waters, 253, 472
 phlegmonous, 457
 etiology of, 457
 pathology of, 458
 symptoms and course of, 458
 treatment of, 459
 polyposa, 461
 silver nitrate in, 268
 subacid, 463
 toxic, 455
 etiology of, 455
 pathology of, 455
 prognosis of, 456
 symptoms of, 456
 treatment of, 456
 with mucus, lavage in, 197, 471
- Gastrochylorrhea, 94, 440
- Gastroenteritis, acute, 635
 from acute gastritis, 453
 infantile, sea-water in treatment of, 642
- Gastroenteroptosis, 557
 Aaron's sign of, 565
 bandages for relief of, 574
 constipation and, 562
 corsets in, 579
 diagnosis of, 564
 etiology of, 557
 fluctuating rib and, 563
 forms of, 557
 hyperalimentation in, 569
 lifting sign of, 565
 massage and exercise in, 574
 neurasthenia and, 562
 point of tenderness in, 564
 pregnancy and, 557, 581
 prognosis of, 569
 prophylaxis of, 569
 symptoms of, 562
 technic of nutrition in, 571
 treatment of, 569
 dietetic, 569
 electrotherapeutic, 574
 hydrotherapeutic, 573
 mechanical, 574
 medicinal, 581
 physical, 574
 surgical, 583
- Gastroenterostomy in gastric ulcer, 507
- Gastrogenic diarrhea, 173, 180, 673.
See Diarrhea, gastrogenic.
- Gastro-intestinal catarrh, furruginous waters in, 254
 crises in intestinal toxemia, 690
 motility, bead test for, 129
 neuroses, treatment of, 391
- Gastroptosis, 557
 pathology of, 558
 roentgenography of, 139
 sea baths in, 257
- Gastrorrhea, 94
 acute intermittent, 440
 chronic, 411. *See* Hypersecretion, continuous.
- Gastroscope, 351, 352
- Gastrosuccorrhea, 94, 440. *See* Hypersecretion, continuous.
- Gastroxynsis, 441
- Gause test for gastric acidity, 89
- Geographic tongue, 323
- Gelatin as a culture medium, 177
 digestibility and food value of, 161
 in gastro-intestinal diseases, 177
 test of digestion and acidity, 84
- Gingivitis, 333
 in pregnancy, 303
- Glands of mouth, 314
 treatment of, 314
- Glass-blowers' oral lesions, 304
- Globon, 192
- Glossitis, acute diffused, 324
 papular, 325
 chronic superficial, 325
 manifested as coating or furring of tongue, 322
 in tertiary syphilis, 312

Glossitis, treatment of, 324
 Glossodynia, 321
 Glutanol, 277
 Gluzinski's test for gastric carcinoma, 88
 Glycerin enemata, 222
 Glycyltryptophan test for gastric carcinoma, 87
 Gout, stomatitis from, 303
 Grape cure of atonic constipation, 664
 Green vegetables in gastric diseases, 167
 Gross's casein test for trypsin, 125
 duodenal tube, 100
 Ground-itch anemia, 806. *See* Hook-worm disease.
 treatment of, 813
 treatment of, 812
 Gruels for gastric patients, 166
 Grutzner-Gamgee test for steapsin in feces, 126
 Gum, inflammation of, 333
 Gum-boil, 331
 Gummata of palate, 311
 Günzberg's test of absorptive power of stomach, 89
 for free HCl in stomach contents, 76

H

HABITUS enteroptoticus, 558
 Hair-tongue, 323
 Half baths, 248
 Handling of intestine, effect of, 783
 Hanot's disease, 596
 Hartenstein's legumins, 193
 Haudek's niche, 141
 Headache from eye-strain, 418
 from hypersecretion, 440
 Heat to abdomen, how applied, 250, 251
 unit for proctoclysis, electric, 241, 242
 gas or alcohol, 243
 value of foods, 151
 Helminthiasis and nervous dyspepsia, 419
 Hemangioma of mouth, 336
 Hematemesis, bisthuth subnitrate in, 266
 following stomach operation, lavage in, 198
 Hematoxylon, 278
 Hemicellulose absorption, 61
 in constipation, 184
 digestion of, 184
 Hemmeter's duodenal apparatus, 98
 Hemocoones in diagnosis, 611
 Hemoglobin agar tubes, 102
 Hemolytic reactions in gastric carcinoma, 543
 Hemorrhage of anal fissure, 861
 differentiation of gastric and duodenal, 512
 esophageal, 383

Hemorrhage from duodenal ulcer, 707, 709
 from gastric ulcer, 512
 from gums in hemophilia, 302
 in purpura, 302
 from rectal polypi, 835
 gastric, 511
 causes of, 511, 512
 diagnosis of, 511
 differential, 512
 from arteriosclerosis, 528
 lavage in, 198, 204, 513
 mortality of, 520
 prophylaxis of, 512
 treatment of, 512
 by analgesics, 518
 atropin, 518
 chloroform water, 519
 orthoform, 519
 by enemata, 514
 by hemostatics, 514
 adrenalin, 271, 517
 bismuth, 517, 520
 blood transfusion, 518
 coagulen, 517
 coagulose, 516
 emetin, 514
 epinephrin, 271, 517
 ergot, 514
 escalin, 518
 gelatin, 515
 "hemostatic serum," 516
 hydrastin, 514
 kephalin, 516
 pituitary extract, 517
 silver nitrate, 518
 stypticin, 515
 thromboplastin, 516
 by lavage, 513
 medicinal, 514, 519
 operative, 520
 hemorrhoidal, 822
 treatment of, 823
 intestinal, 511, 747
 diet in, 172
 enemata in, 514
 iodin in, 520
 treatment of. *See* Hemorrhage, gastric.
 into bile ducts, 614
 occult, in duodenal ulcer, 709
 of pancreas, 631
 Hemorrhoidal electrode, Williams', 832
 pessaries, 826
 Hemorrhoids, 821
 age and sex incidence of, 822
 anatomy of, 821
 as a cause of nervous dyspepsia, 419
 cleanliness in, 823
 complications of, 822
 constipation as a cause of, 821

- Hemorrhoids**, development of, 821
 diet in, 823
 etiology of, 821
 external, 821
 hemorrhage from, 822
 treatment of, 823
 internal, 821
 pain of, ice-bag (Zweig's) for, 824
 leeches for, 825
 pessaries for, 825, 826
 symptoms of, 822
 treatment of, 822
 bloodless, 827
 by chrysarobin suppositories, 825
 by electrolysis, 831
 extra-anal, 827
 injection, 828
 mineral water, 225, 823
 phenol, 828
 quinin and urea hydrochlorid, 825
 sphincter stretching (Verneuil's), 827
 surgical, 832
 Terrell's, 829
- Hepatic cirrhosis**, 590 *See* Cirrhosis.
 insufficiency, levulose test for, 593.
See also Liver.
 lipase test for, 592
 phthalein test for, 593
 urobilin test for, 593
- Hepatitis**, 584
 amebic, emetin treatment of, 723
 suppurative, 584
 ipecac in prevention of, 723
- Hepatoptosis**, 568, 605
 treatment of, 605
- Hernia**, epigastric, as a cause of nervous dyspepsia, 419
 epigastrica, 555
 recti, 845
- Herpes zoster** in esophagus, 357
 in mouth, 318
- Hexosane**, a hemucellulose, 61
- Hiccough** (singultus gastricus), 408
 treatment of, 408
- High colonic irrigation**, 221
- High-frequency current** in digestive disorders, 219
- Hill's esophagoscope**, 352
 gastroscope, 352
- Hirschman's anoscope**, 830
 rectal massage treatment of chronic constipation, 229
- Hirschsprung's disease**, 700
 Roentgen-ray diagnosis of, 148
- Honthin**, 277
- Hookworm**, course of, to intestine, 810
 disease, 806
 in Africa, 808
 anemia of, 813
 carriers of, 809
 castor oil in treatment of, 813
 diagnosis of, 811
- Hookworm disease**, distribution of, 806
 eosinophilia in, 811
 leukopenia in, 811
 pathology of, 810
 poikilocytosis in, 810
 polychromatophilia in, 810
 retarded development in, 811
 in South, 806
 symptoms of, 811
 treatment of, 812
 with chenopodium, 812
 with eucalyptus, 813
 with male-fern, 813
 with salicylic acid, 812
 with thymol, 812
 multiplication of the, 809
 penetration of skin by, 810
- Hormonal** in atonic constipation, 667
- Hormones**, 55, 667
- Hot applications**, 250
- Hour-glass stomach**, 142, 524
 pseudo, 139
 shown by Roentgen ray, 142
- Hunger**, abnormal (bulimia), 415
 contractions of stomach, 64
 pain, 491, 706, 708
- Hydratic and thermic treatment**, 247
- Hydrochloric acid**, action of, 54
 in pancreatic affections, 259, 627
 administration of, 260
 Benedict's effervescence test for, 88
 bile secretion and, 259
 capsule method of taking, 260
 in chronic gastritis, 258, 469
 combined, in stomach contents, 81
 alizarin test for, 81
 effect of, on pylorus, 259
 free, after Ewald-Boas breakfast, 77
 Riegel dinner, 77
 and combined, phenolphthalein test for, 79
 dimethylamidoazobenzol test for, 75, 89
 Günzburg's test for, 76
 normal solutions in titrating, 77
 Töpfer's test for, 80
 Friedrich's test for, 89
 in gastric diseases, 258
 ulcer, 499
 in gastrogenic diarrhea, 674
 gauze test for, 89
 in hyperchlorhydria, 436
 medicinal effects of, 258, 436
 pancreatic function and, 259
 pepsin and, 259
 pepsinogen and, 83
 proteolysis and, 259
 secretion in anomalies of menstruation, 85
 in appendicitis, 8^a

- Hydrochloric acid secretion in chole-
 lithiasis, 85
 in chronic gastritis, 85
 in nervous dyspepsia, 85
 in stomach contents, tests for,
 75
- Hydrocyanic acid, dilute, as a gastric
 sedative, 269
- Hydrogen peroxid in gastric diseases,
 272, 436
- Hydrops of gall bladder, 616
- Hydrotherapeutics, 247
 in atonic constipation, 665
 in gastroenteroptosis, 573
 indications for, 249
 in membranous enteritis, 656
 in nervous diarrhea, 681
- Hygiama, 193
- Hymenolepis nana, 798
- Hyperacidity, 94. *See also* Hyper-
 chlorhydria.
 alkaline carbonated waters in, 254
 atropin in, 271
 Carlsbad water in, 253
 menstruation and, 419
 test-diet stool findings in, 131
 with diarrhea, 438
 with pain, 439
- Hyperalimentation, 570
 in gastroenteroptosis, 569
 in mucous colitis, 653
 muscular exercise and, 571
 in spastic constipation, 671
- Hyperchlorhydria, 94, 430
 carbohydrate digestion in, 85
 diagnosis of, 431
 etiology of, 430
 hyperacidity of, 438, 439
 pathology of, 431
 physiotherapeutic measures in, 438
 prognosis of, 432
 symptoms of, 431
 treatment of, dietetic, 422
 fats and oils in, 434
 hygienic, 432
 lavage, 438
 medicinal, 435
 acids, 436
 alkalis and alkaloids, 436,
 437, 438
 analgesics, 436
 astringents, 435
 atropin, 435
 peroxides, 436
- Hyperemia of liver, 588
- Hyperesthesia of esophagus, 384
 of stomach, 413
- Hyperkinesia, 392
- Hypermotility of stomach, 392
- Hyperorexia, 415
- Hypersecretion, 91, 446
 alimentary, 447
 diagnosis of, 447
 eumydrin in, 271
 symptoms of, 447
- Hypersecretion, alimentary, treatment
 of, 448
 by lavage, 448
 medicinal, 448
- alkaline carbonated waters in, 254
- continuous, 441
 diagnosis of, 443
 by external examination
 of stomach, 443
 etiology of, 442
 prognosis of, 443
 symptoms of, 442
 treatment of, 443
 dietetic, 444
 by lavage, 446
 medicinal, 445
 by mineral waters, 447
 physical, 447
 surgical, 447
- from vagotonia, 389
- intermittent, 440
 diagnosis of, 441
 etiology of, 440
 symptoms of, 440
 treatment of, 441
- periodic, 440
 stomach contents in, 94
- Hypertrophy, muscular, of esophagus,
 362
- Hypodermic solutions in ampoules, 581
- I
- ICHTHYOL in intestinal diseases, 281
- Ileal regurgitation, 684
 stasis, roentgenographic diagnosis,
 of, 144
- Ileocecal valve, insufficiency of, 783
- Ileum, carcinoma of, 764
 kink of, 559
 stasis of, 144
 stricture of, 756
- Ileus, 742
 atropin in, 754
 chloroform water irrigations in, 227
 diagnosis of, 745
 external, 742
 from adhesions, 743
 from angulations, etc., 742
 from arteriomesenteric con-
 traction, 743
 from compression, 743
 internal, 743
 from ascariides, 774
 from calculi, 743
 from fecal tumors, 744
 from foreign bodies, 744
 from invagination, 743
 from stricture, 743
 from torsion, 742
 opium in, 753
 paralytic, 744
 puncture in, 753
 spastic, 744

- Ileus, symptoms of, 745
 toxemia of, 747
 treatment of, 747
 by insufflation of air, 752
 internal, 749
 by lavage, 750
 medicinal, 753
 nutritional, 750
 by rectal injections, 741
 irrigation, 752
 surgical, 748
 Incisura of stomach in gastric ulcer, 139
 Incubator test for fecal fermentation and putrefaction, 116
 Indican in urine, 686
 Indol in the intestine, 686
 Infection, focal, 290
 Influenzal stomatitis, 302
 Innervation, contrary, 65
 Insufflation of air in intussusception, 228, 752
 Intestinal absorption, 60, 61
 adhesions, mud baths in, 287
 anastalsis, 63
 antifermentatives, 279
 antiperistalsis, 63
 antiseptics, oxygen for, 105
 antiseptics, 279
 astringents, 276
 atony, massage in, 208
 bacteria, growth of, 686
 Metchnikoff's tests with, 685
 volume of, 686
 carcinoma, 761. *See* Carcinoma of intestine.
 catarrh, acute, 635
 diagnosis of, 637
 etiology of, 635
 feces in, 637
 from cold, 635
 from food, 635
 from intoxication, 635
 infectious, 635
 of large or small intestine, distinction between, 637
 pathology of, 635
 prognosis of, 638
 symptoms of, 636
 treatment of, 638
 dietetic, 172, 639
 dry, 237
 irrigation, 232
 medicinal, 641, 643
 sea-water, 642
 chronic, diagnosis of, 645
 pathology of, 644
 prognosis of, 647
 symptoms of, 644
 test-diet stool findings in, 131, 132, 646
 treatment of, 647
 with constipation, 650
 agar in, 650
 Intestinal catarrh, chronic, with constipation, diet in, 650
 ionization in, 651
 liquid petrolatum in, 650
 rest in, 650
 with diarrhea, 647
 bismuth in, 649
 calcium in, 648, 649
 diet in, 647, 648
 gelatin injections in, 649
 kaolin in, 649
 mineral waters in, 254, 649
 rest in, 647
 vaccine in, 649
 with hyperacidity, 650, 651
 with pain and tenesmus, 651
 dry treatment of, 237
 See also Intestinal irritation.
 colic, 783. *See* Enteralgia.
 contents, normal movement of, 64
 digestion, 56, 61
 diseases, diet in, 172
 electricity in, 230
 location of, by test-diet, 173
 milk in diet of, 174
 mineral waters in, 254, 649
 opium in, 274
 purgatives in, 282
 rectal massage in, 229
 treatment of, mechanical, 228
 through the rectum, 220
 uzara in, 276
 displacements and constipation, 696
 douche, 228
 dyspepsia, oxygen insufflation in, 105
 fermentation and putrefaction, differentiation of, 173
 fermentative dyspepsia, 181
 diet in, 181
 oxygen by the duodenal tube in, 679
 test-diet stools in, 134
 treatment of, 679
 function, test diet to determine, 112
 gases, origin of, 699
 hemorrhage, 511
 intussusception, 743
 irrigation, 232
 antiseptic, 235
 astringent, 236
 heating apparatus used in, 240
 sedative, 236
 technic of, 233
 with mineral waters, 237
 irritation, pathologic effects of, 685
 treatment of, 232
 dry, 237
 juice, 59

- Intestinal juice, calcium carbonate in, 59
 ferments in, 59
 variation in consistency of, 59
 kinking, 561
 and colonic dilatation, 791
 lesions in hookworm disease, 810
 lipoma, 836
 treatment of, 836
 lymphosarcoma, 766
 pathology of, 766
 symptoms of, 766
 treatment of, 766
 massage, 211
 motility, carmin and charcoal tests of, 128
 Einhorn's bead test of, 129
 movements, 62
 nerve control of, 63, 64
 pendulum, 63
 peristaltic, 62
 segmenting, 62
 myoma, 836
 treatment of, 836
 neoplasms, benign, 766
 constipating diet in, 172
 neuroses, 781
 high-frequency currents in, 219
 obstruction, 742. *See also* Ileus.
 an early manifestation of, 745
 enemata to remove, 227
 etiology of, 742
 roentgenography in, 149
 symptoms of, 745
 treatment of, 747
 occlusion, 742. *See* Ileus.
 organs of perception, 56
 pain, 831. *See* Enteralgia.
 papillæ, 836
 treatment of, 837
 parasites, 794
 paresis, 782
 causes of, 783
 diagnosis of, 783
 following operation, lavage in, 197
 pituitrin in, 284, 703
 treatment of, 783
 perforation by round worm, 801
 peristalsis, 62
 effect of opium on, 275
 relation of nodal tissue to, 63, 562, 696
 strychnin as an aid to, 284
 polypi, 835
 radical removal of, 835
 protectives, 276, 279
 putrefaction, 687. *See also* Intestinal toxemia.
 beverages in, 180
 combined indolic and saccharobutyric type of, 688
 diet in, 174, 178
 indolic type of, 686, 687
 Intestinal putrefaction, saccharobutyric type of, 687
 toxic products of, 686
 reflexes, chemical and motor, 57
 restlessness, 782
 sarcoma, 766, 834
 pathology of, 766
 symptoms of, 766
 treatment of, 766
 sedatives, 274
 spasm, 781
 appearance of stools in, 781
 treatment of, 781
 stasis, 683
 duodenal lavage in, 107
 oral sepsis and, 290
 petroleum jelly in, 694
 physiology of, 683
 pyorrhea and, 260
 treatment of, 689
 surgical, 697
 stenosis, enemata in, 227
 insufflation of air in, 228
 roentgenographic diagnosis of, 148
 strictures, 755. *See* Stricture.
 lavage in, 759
 tonus, 64
 toxemia, 683
 abdominal bandages in, 696
 antiseptic diet in, 689
 buttermilk, 690
 carbohydrates, 690
 sour milk, 690
 whey, 690
 medication in, 692
 bacterial growth in, 684, 686
 betanaphthol in, 693
 chloramine-T in, 693
 constipation of, 694
 course of, 685
 diagnosis of, 686
 duodenal lavage in, 695
 etiology of, 683, 684
 hexamethylenamin in, 693
 ichthyol in, 693
 indicanuria in, 686
 lavage in, 695
 symptoms of, 688
 treatment of, 689
 anticonstipation, 694
 antiseptic, 689, 692
 bacterial, 691
 dietetic, 689
 lavage, 695
 medicinal, 692
 surgical, 696
 tuberculosis, 737, 740
 diagnosis of, 738
 prognosis of, 738
 test-diet stool findings in, 133
 treatment of, 739, 741
 dietary, 739
 medicinal, 739
 specific, 740

- Intestinal tuberculosis, treatment of,**
 asymptomatic, 739, 740
 tumors, 172, 761, 766
 ulcers, 705, 713, 719, 730, 737
See Ulcer, duodenal, and Ulcers, intestinal
 wall, pathologic products of, 121
Intestine—effect of banding, 783
 pendulum movements of, 63
 Roentgen-ray examination of, 143
 segmenting movements of, 62
 short-circuiting the, 697
 small, relative importance of, in intestinal diseases, 180
Intra-intestinal medication, dry, 237
 powder blowers, 238
Intrarectal treatment of intestinal diseases, 220
Intraventricular electrization, 216
Intussusception, 743
 hemorrhage from, 747
 incidence of, 643
 insufflation of air in, 228, 752
 rectal irrigations for, 228
Invertin 59
Iodid salivation, 301
Iodin germs, 181
 in gastric diseases, 272
Iodoform in diseases of mouth, 297
 mass, preparation of, 298
Ionization in enteritis, 651
Iron, organic 188
Irrigating fluid for duodenal lavage, 106
 tubes, 233, 234
Irrigation of intestine, 227, 232. *See* also Intestinal irrigation.
 of stomach, 205
Itching of anus, 851
- J**
- JAUNDICE,** febrile, 587. *See* also Cholangitis and Cholecystitis.
 serum treatment of, 588
 Carlsbad waters in, 611
 catarrhal, 607
 development of, 609
 functional, 609
 hematogenous, 609
 hepatic cysts and, 604
 itching and, 609
 sequelae of, 609
Jejunal carcinoma, 764
 stricture, 756
 ulcer, 712
Jutte's apparatus for duodenal lavage, 103
 duodenal tube, 100
- K**
- KARLI milk-cure** of dropsy, 163
 Kefir in diet, 164
 Kefir, koumiss, yoghurt and sour milk, comparison of, 165
 Keith's nodal tissue, 63, 561, 696
 Kelly's proctoscope, 230
 Kidney, movable, 566
 chronic appendicitis and, 768
 palpation of, 566
 Kinases in digestion, 53
 Kinking, intestinal, 561
 Koumiss in diet, 164
 Kuhn's duodenal tube, 98
- L**
- LACERATIONS,** obstetric, nervous dyspepsia and, 419
 Lacing gastroenteroptosis and, 557
 Lacrimal glands, enlargement of, 330
 Lactase, 59
 Lactic acid in intestinal diseases, 279
 in stomach contents, 82
 Strauss' test for, 83
 Uffelmann's test for, 82
Laennec's disease, 590
Lane's short-circuiting operation, 697
Lavage, 197
 in achylia gastrica, 471
 in alimentary hypersecretion, 448
 in colonic carcinoma, 766
 in continuous hypersecretion, 446
 in convulsions from overeating, 198
 in diabetes mellitus, 198
 duodenal, 105. *See* also Duodenal lavage.
 in eclampsia, 198
 in enteritis membranacea, 655
 ulcerative, 733
 in gastric atony, 476, 482
 dilatation, 197, 482, 487
 relaxation, 208
 tetany, 198
 gastric or colonic, apparatus for, 199
 in gastritis, acute, 198, 450, 454
 chronic, 471
 with mucus, 197, 471
 in gastrogenic diarrhea, 677
 in hematemesis, 198
 in hemorrhage from gastric ulcer, 198, 513
 in hyperchlorhydria, 438
 in hypersecretion, 448
 in ileus, 750
 in intestinal catarrh, 649
 paresis, 197
 stricture, 759
 toxemia, 695
 medicated, in nervous gastralgia, 210
 in meteorism of typhoid, 198
 in motor insufficiency, 476, 482
 in nephritis, 198

- Lavage** by patient, 201. *See also* Auto-lavage
 in poisoning, 197
 preceding gastric or intestinal surgery, 197
 for prevention of vomiting, 197
 in pyloric stenosis, 197, 198
 for removal of gastric mucus, 197, 205, 471
 of stomach, 197
 apparatus for, 198, 199
 contra-indications against, 198, 204
 duration of, 204
 indications for, 197
 safety of, 203
 technic of, 199, 200
 time for giving, 204
 in vomiting, 197
 of peritonitis, 198
 postoperative, 197
 with Aaron's improved stomach tube, 71
 without the stomach tube, 201
- Law** of contrary innervation, 65
- Laxative** diet, 182
 drugs, 284
 agar, 662
 inorganic salts, 284
 liquid petrolatum, 664
 phenolphthalein, 286
- Lead** stomatitis, 301
- Leather-bottle** stomach, 546
- Legumes** in diet of gastric patients, 167
- Legumins**, Hartenstein's, 193
- Lester's** coiled tubing, 251
- Lenhart's** treatment of gastric ulcer, 498
- Leontiasis** osses, 345
- Leprosy** of mouth, 314
 treatment of, 315
- Leube's** diverticular sound, 376
 test meal for testing gastric motility, 90
- Leube-Ziemssen** treatment of gastric ulcer, 495
- Leukocytosis** in acute appendicitis, 775
- Leukopenia** in hookworm disease, 511
- Leukoplakia**, 319
 from glass-blowing, 304
 mouth-washes in, 320
 oral carcinoma and, 342
 radium in treatment of, 320
 removal of plaques of, 320
- Levulose** in urine, test for, 363
- Leyden** and Ranvier's esophageal bougie and cannula, 371
- Lichen** planus in mouth, 317
 treatment of, 317
- Lingua** geographic, 325
 macra, 325
 papata, 326
- Lingual** abscess, 324
 phlegmon, 324
 toxic, hyperemias of, 327
 hypertrophy of, 326
- Lingual** tonsillitis, acute, 326
 ulcers, 325
- Linitis plastica** hypertrophica, 546
- Linseed** poultices, 250
- Liparin**, 194
- Lipase**, 56
 tests for, 592
- Lipoma** of esophagus, 362
 of intestine, 836
 of mouth, 335
 of rectum, 836
 of stomach, 555
- Lips**, abscess of, 334
 carcinoma of, 342
 congenital fistulae of, 333
 eczema of, 317
 treatment of, 317
 exfoliative inflammation of, 334
 inflammation of, 334
 acute, 334
 chronic, 334
- Liquid** petrolatum, laxative action of, 664
 in rectal carcinoma, 834
- Liver**. *See also* Febrilis icterus, Cirrhosis, and Hepatic insufficiency.
 abscess of, 584
 symptoms of, 585
 treatment of, 586
 acute affections of, 584
 angoma of, 602
 atrophy of, acute yellow, 586
 treatment of, 587
 brown, 598
 partial, 598
 red, 598
 carcinoma of, 600
 chronic affections of, 588
 cirrhosis of, 590. *See* Cirrhosis of liver.
 cysts of, 602
 diseases of, 584
 dislocation of, 588
 echinococci in, 602
 diagnosis of, 603
 symptoms of, 603
 treatment of, 604
 fatty, 604
 etiology of, 604
 pathology of, 605
 treatment of, 605
 fibrosis of, 602
 floating, 606
 function, tests for, 592, 593
 hyperemia of, active, 588
 diagnosis of, 589
 treatment of, 589
 passive, 589
 symptoms of, 589
 treatment of, 590
 inflammation of, 584
 treatment of, 584
 neoplasms of, 600
 neurlgia of, 606
 parasites of, 602

- Liver**, roentgenography in study of, 149
 sarcoma of, 601
 syphilis of, acquired, 599
 diagnosis of, 600
 symptoms of, 599
 treatment of, 600
 congenital, 599
 tumor from echinococci, 604
Loewi's pupillary symptom of pancreatic insufficiency, 626
Ludwig's angina, 330
 treatment of, 331
Lupus erythematosus in mouth, 317
 of oral mucosa, 312
Lymph glands, nodiform and verrucous, in mouth, 337
Lymphangioma of mouth, 337
Lymphoma, cystic, of mouth, 337
 diffuse, of mouth, 337
Lymphosarcoma of intestine, 766
- M**
- MACMILLAN'S** rectal tampon treatment of chronic constipation, 230
Macroglossia, 326
Maggot worm, 803
Magnesium peroxid in gastric diseases, 272
 sulphate for draining gall bladder and bile ducts, 105, 612
 subcutaneously, 666
Malar mucosa, affections of, 334
 carcinoma of, 344
Malformation of tongue, 322
Malignant growths, intestinal, 761.
 See also Carcinoma and Sarcoma.
 constipating diet in, 172
Malleus in man, 313
Maltase, 59
Maltose, 50
Mammala, 192
Mandrake as a cathartic, 286
Marasmus and duodenal ulcer, 710
Massage, 208
 abdominal, 212, 214
 in atonic constipation, 208, 664
 in connective-tissue adhesions, 208
 in gastric dilatation, 208, 483
 relaxation, 208
 retention, 208
 in gastritis, chronic, 472
 in gastroenteroptosis, 574
 in ileus, 753
 in intestinal atony, 208
 of intestine, 211
 technic of, 211-213
 and medicated lavage in nervous gastralgia, 210
 in motor insufficiency, 477, 483
 in nervous dyspepsia, 208, 423
 in pyloric stenosis with dilatation, 208
Massage in pylorospasm, 209
 rectal, in chronic constipation, 229
 in relaxation of stomach, 208
 in retention of gastric contents, 208
 of stomach, 208
 contra-indications for, 208
 indications for, 208
 technic of, 209
 of sympathetic nerve plexuses, 214
 vibratory, 211
 in volvulus, 749
Matthews' treatment of ulcerative colitis, 733
Maxilla, adamantoma of, 345
 carcinoma of, 346
 chondroma of, 345
 cysts of, 345
 odontoma of, 345
 osteoma of, 345
 sarcoma of, 346
Meal. *See* Test meal.
Measles, diseases of salivary glands and, 329
 oral manifestations of, 301
Meat and gastric secretion, 53
 composition of, 160
 in diet, 159
 digestibility of, 161
 extracts, 194
 composition and relative values of, 195
 preparation of, 160
 restriction of, 160
Meats, canned, 160
 in constipating diet of intestinal diseases, 179
 light and dark, 159
 raw, rare and smoked, in gastric diseases, 160

 salted, 160
Mechanical treatment of intestinal diseases, 228
Meckel's diverticulum, 742
Medication in gastric diseases, 258
 in intestinal diseases, 274
Medicinal exanthems, 301
Megacolon, congenital, 790
Melena in infants, relation of duodenal ulcer to, 710
Meltzer's sign of acute appendicitis, 770
Menstrual anomalies, HCl secretion in, 85
 nervous dyspepsia and, 419
Menstruation, gastric acidity and, 419
Mercurial stomatitis, 299
Merycism, 405
Metagen, 159
Metchnikoff on Bulgarian milk, 165
Metchnikoff's tests with intestinal bacteria, 685
Meteorism, 698
 hormonal in, 667, 703
 large enemata in, 228
 physostigmin in, 703

- Meteoriam, pituitary extract in, 703
Metric weights and measures, 862
Microscopic examination of stomach contents, 92
Mikulicz's disease, 330
Milk, boiled, for young patients, 176
 Bulgarian, 165
 in catarrh of small intestine, 176
 compared with artificial foods, 189
 cure of dropsy, 163
 in diet, 162, 175
 in gastric diseases, 175
 gastric secretion and, 53
 in intestinal diseases, 174, 175
 modification of, 163
 by boiling, 176
 by lime water, 163
 by salicylic acid, 176, 178
 preparations, 176
 Gartner's fat milk, 194
 kefir, 194
 koumiss, 194
 vegetal le milk, 194
 Volmer's mother's milk, 194
 yoghurt, 164
 protein preparations, 191
 Schmidt's method of reducing the irritating effects of, 176
 somatose, 192, 277
 sour, kefir, etc., 165
 sugar, fermentation of, in the bowel, 175
 vegetable, 194
Mineral baths, 256
 radio-activity of, 257
 oil as a laxative, 582, 664
 water cures, 254, 255
 waters, 168, 247, 252, 582. *See* also Carlsbad.
 in acute gastritis, 253
 alkaline carbonate, 253
 chlorin, 252
 litter, 254
 mode of action of, 255
 in chronic gastritis, 255
 gastrorrhoea, 447
 intestinal catarrh, 254, 649
 ferruginous, 254
 in chronic diarrhoea, 256
 in gastric diseases, 167
 in motor insufficiency, 477, 483
 purgative, action of, 254
 constipating effect of, 255
 time for taking, 254
 sodium chlorid, 253
 effect of, on gastric secretion, 255
Miner's anemia, 546
Mastagmin reaction in gastric carcinoma, 544
Masterson, 742
Mixed nutritive preparations, 196
Morgan's modification of Einhorn's chondral alimentation, 362
Morphism as an intestinal sedative, 274
Motility of colon, roentgenographic study of, 145
 of intestine shown by bead test, 129
 by carmin or charcoal, 129
 of stomach, disturbance of, 140.
 See Motor function.
 shown by Roentgen ray, 140
Motor function of stomach, 50, 90
 chlorophyl test of, 91
 Einhorn's bead test of, 129
 Leube's test meal and, 90
insufficiency, 95, 473
 of first degree (atony), 473
 diagnosis of, 474
 etiology of, 473
 symptoms of, 474
 treatment of, 475
 dietetic, 475
 by lavage, 476, 482
 medicinal, 477
 physical, 477
 with mineral waters, 477
 of second degree (dilatation), 478
 bandages in, 483
 diagnosis of, 480
 drugs in, 483
 etiology of, 478
 galvanism in, 483
 lavage in, 482
 massage in, 483
 mineral waters in, 483
 rectal alimentation in, 482
 subcutaneous nutrition in, 482
 symptoms of, 479
 thirst of, 481
 treatment of, 481
 shown by food remnants in the stomach, 95
neuroses, 387
 treatment of, 391
Mould fungi in the stomach, 92
Mouth, actinomycosis of, 316
 adenoma of, 540
 anatomy of, 266
 angewarts of, 536
 bacteria in, 289, 700
 berms, 313, 314
 carcinoma of, 542
 relation of leucoplakia to, 342
 cysts of, 539, 557, 558, 559
 diseases, 289
 calcification in, 286
 in constitutional infectious diseases, 317
 non-infectious diseases, 312

- Mouth diseases, feeding in, 298**
 from bismuth, 300
 from bromids, 301
 from general intoxications, 299
 from iodids, 301
 from lead, 301
 from mercury, 299
 prophylaxis and treatment of, 300
 from silver, 301
 from yellow phosphorus, 301
 general treatment of, 293
 in glass-blowers, 304
 iodoform in, 297
 local anesthetics in, 299
 nutrition of patients with, 298
 occupation and, 295
 potassium chlorate in, 297
 secretory, 321
 trophic, 321
 vasomotor, 321
 endothelioma of, 340
 erosions, 303
 etiology and treatment of, 303
 exanthems, 301
 fibroma of, 335
 glanders of, 313
 hemangioma of, 336
 herpes of, 318
 hygiene of, 293
 leprosy of, 314
 lichen planus of, 317
 lipoma of, 335
 lupus of, 312
 erythematosus in, 317
 lymphangioma of, 337
 lymphoma of, 337
 manifestation of measles in, 301
 myxoma of, 336
 nervous affections of, 320
 papilloma of, 340
 parasites in, 320
 pemphigus of, 317
 phlegmons of, 324, 330
 ranula of, 339
 in scarlet fever, 301
 scleroma of, 315
 skin diseases in, 316
 sore, in chlorosis, 302
 strumæ of, 339
 syphilis of, 309
 primary, 309
 secondary, 310
 tertiary, 311
 telangiectasia of, 336
 tuberculosis of, 312
 tumors of, benign, 335, 345
 malignant, 341, 346
 typhoid ulcers in, 302
 urticaria in, 318
 vaccinia in, 302
 varicella in, 302
 variola in, 302
Mouth-washes in gangrenous stomatitis, 306
- Mouth-washes in leukoplakia, 320**
 recommended, 295, 296
Movable kidney, appendicitis and, 768
 palpation of, 566
Mucous colitis, 652
 membrane in stomach contents, 94
Mucus from small and large intestine, differentiation of, 121
 in stomach, lavage for removal of, 197
 in stool, 121
Mud baths in gastric neuroses, 257
 in intestinal adhesions, 257
Muller-Schlecht test for trypsin, 125
Murphy drip, 239
 in acute appendicitis, 774, 778
 in duodenal feeding, 502
 in severe intestinal catarrh, 641
Murphy's sign of cholelithiasis, 619
Muscle remnants in feces, 120
Muscular exercise and hyperalimentation, 571
 relaxation of stomach, massage in, 208
Mutase, 191
Myasthenia gastrica, 473
Myer's cardia dilator, 397
Myiasis intestinalis, 820
Myoma of esophagus, 362
 of rectum, 836
Myxoma in mouth, 336
Myxosarcoma, maxillary, 346
- N**
- NARCOTICS in intestinal diseases, 274**
Nasal feeding, 299
Naunyn's sign of cholelithiasis, 619
Nausea, nervous, 415
 treatment of, 415
Necator americanus, 806
Necrosis of pancreas, 631
Neosarsphenamine, 534
Neoplasms of bile ducts and gall bladder, 614
 of esophagus, 362
 of intestine, 761, 766
 of liver, 600, 610
 of mouth, 336
 of stomach, 537, 553. *See Stomach.*
Nephritis, gastric lavage in, 198
Nephroptosis, 565
Nerve, facial, paralysis of, 320
 plexuses accessible by massage, 214
 of intestine, 64
 supply of stomach, 52
Nervous affections of intestine, 392, 781
 of mouth, 320
 of stomach, 392
 anorexia, 418
 diarrhea, test-diet stools in, 134, 681

- Nervous dyspepsia, 418. *See* Dyspepsia, nervous.
 eructation, 401. *See* Aerophagy.
 nausea, 415
 system, autonomic, 388
 vegetative, 387, 390
 vomiting, 403
 Neuralgia of liver, 605
 of stomach, 410
 of tongue, 321
 Neurasthenia gastrica, 418
 gastroenteroptosis and, 562
 Neuritis from lack of vitamin, 157
 Neuroses of esophagus, 384
 of intestine, 781
 motor, 387
 sea baths in, 257
 secretory, 430, 440
 sensory, 410
 of stomach, 387
 and intestine, treatment of, 391
 electricity in, 215
 eye-strain and, 418
 gastric contents in, 94
 Nicotin in gastric diseases, 271
 Nitroglycerin as a gastric sedative, 269
 Nodal tissue in intestine, 64, 561
 Nodes, peristaltic, 562, 696
 Noma, 306
 treatment of, 307
 Normal solutions in quantitative analysis of stomach contents, 77
 Nothnagel's contribution to fecal examination, 112
 Nuclei, effect of pepsin, trypsin and crepsin upon, 126
 in feces, significance of, 126
 pancreatic digestion of, 624
 test of Adolf Schmidt, 126
 Nurslings and oral antiseptics, 297
 Nutrient enemata, 243
 Nutrition of patients with mouth diseases, 298
 subcutaneous, 482
 technic of, in gastroenteroptosis, 571
 Nutritive preparations. *See* Food preparations.
 Nutritive-Heyden, 192
 Nutrolo, 194
 Nutrose, 191
 Occupation and mouth diseases, 295
 Ochsner's method of treating acute appendicitis, 774
 Odda, 193
 Odontoma of maxilla, 345
 Oil enemata, 223
 enemator, Roberts', 225
 Zweig's, 224
 Olive oil agar tubes, 102
 in gastric diseases, 273
 Onions in gastric diseases, 166
 Opium as an evacuant, 283
 in ileus, 753
 in intestinal diseases, 274, 275
 preparations in intestinal disease, 275
 Oral antiseptics for nurslings, 297
 effects of antipyrin, quinin, phenacetin, and acetylsalicylic acid, 301
 of influenza, 302
 of measles, 301
 of paratyphoid fever, 302
 of rubeola, 302
 of scarlet fever, 301
 of typhoid fever, 302
 of vaccinia, 302
 of varicella, 302
 of variola, 302
 lesions, traumatic, 304
 treatment of, 304
 lupus, 312
 local treatment of, 313
 sepsis, 290
 constitutional diseases and, 291
 duodenal ulcer and, 706
 gastric ulcer and, 489
 organic infections and, 292
 See also Mouth.
 Orexin in gastric diseases, 267
 Orthoform-new as a gastric anodyne, 270
 Osteoma of maxilla, 345
 of mouth, 336
 Otitis from parotitis, 329
 Oxygen insufflation as an intestinal antiseptic, 105, 679
 in intestinal fermentative dyspepsia, 679
 Oxyuris vermicularis, 803, 804. *See also* Thread worm.

O

- Obstruction of common bile duct,
 signs of, 109
 of intestine, 742
 xerontgenography in, 149
 Occult blood in feces, benzidin test for, 123
 phenolphthalein ring test for, 124
 in intestinal tuberculosis, 738

P

- Packs, cold, 248
 warm, 249
 Pain of acute gastritis, 454
 of duodenal ulcer, 706, 707, 708
 gastric, 266, 270. *See* Gastralgia.
 of gastric erosions, 522
 hyperesthesia, 413
 ulcer, 490, 491
 of hemorrhoids, 824

- Pain, hunger, 491, 706, 708**
 of hypersecretion, 442
 of ileus, 745
 of intestinal obstruction, 745
 ulcer, 706
 sign of appendicitis, acute, 770
 chronic, 772, 773
 of pancreatic disease, 627
- Pains in chronic intestinal catarrh with diarrhea, 645**
 dysenteric, epinephrin in, 726
 local and internal treatment for, 726
 of sarcoma, 553
- Palatal carcinoma, 344**
 effects of rubeola, 302
 gummata, 311
 ulcers, 304
 in typhoid fever, 302
- Palefski's duodenal tube, 100**
- Pancreas, diseases of, 623. See Pancreatic and Pancreatitis.**
 inflammation of, 623. *See* Pancreatitis.
 roentgenography in study of, 149
- Pancreatic affections, action of hydrochloric acid in, 259, 627**
 diet in, 627, 629
 internal treatment of, 627, 629
- calculi, 633**
carcinoma, roentgenography and, 149
cysts, 632
 laparotomy in, 632
 roentgenography in, 149
 digestion of nuclei, 624
 ferments, 57
 fistula, antidiabetic diet in, 630
 surgery in, 631
 function, duodenal contents and, 120
 HCl and, 259
 Sahli's glutoid test of, 129
 hemorrhage, 631
 infantilism, 634
 insufficiency, epinephrin test of, 626
 pupillary symptoms of, 626
 juice, 57
 necrosis, 631
 obstruction, pilocarpin in, 630
 preparations, 263
 secretion, 57
 anomalies of, 103
 determination of, 101, 102, 103
 effect of carbohydrates and proteins on, 629
 of hydrochloric acid on, 259
 tumors, 633
- Pancreatin, 262**
 digestive properties of, 262
 preparations in chronic pancreatitis, 628
- Pancreatitis, acute, 628**
 Pancreatitis, acute, percussion sign of, 629
 treatment of, 628
 chronic, 623
 carbohydrates in, 628
 diagnosis of, 624
 by Cammidge test, 626
 by fat test, 625
 by Loewi's test, 626
 by oil test breakfast, 626
 by percussion, 629
 by protein test, 624
 by starch test, 625
 etiology of, 624
 pathology of, 624
 prognosis of, 627
 symptoms of, 623
 test-diet stool in, 624
 treatment of, 627
 dietetic, 627
 surgical, 628
 use of duodenal tube in, 625
 duodenal contents in, 109
 pancreatic secretion and, 109
- Pancreon, 263**
Pantopon, 275
Papaverin as an antispastic, 275
Papayotin, 263
Papillæ of rectum, 836
Papilloma in esophagus, 362
 of mouth, 340
 mucous, 555
- Paracentesis in ascites, 595**
Paraffin enemata, 225
Paralysis of esophagus, 385
 of facial nerve, 320
 of rectum, 849
- Parasites of bile ducts, 616**
 in esophagus, 361
 of intestine, 794
 of liver, 602
 in mouth, 320
- Paratyphilitis, 767**
Paratyphoid fever, palatal ulceration in, 302
 Paresis of intestine, 849
 and paralysis of rectum, 849
- Parotitis, epidemic, 329**
 treatment of, 329
 ulcerous, 329
- Parulis, 331**
Passio iliaca, 742. See Ileus.
Pathogenesis of gastric ulcer, cholecystitis, etc., 292
Pathologic changes reflected in gastric secretion, 94
 food remnants, 119
 products of the intestinal wall, 121
 stools and their significance, 119
- Pawlow's experiments in digestion, 52, 54, 56**
Peas in the diet of gastric patients, 167
Peguin, 163
Pemphigus in esophagus, 357
 of oral mucosa, 317

- Pendulum movements of intestine, 63
 Pentosane, a hemicellulose, 61
 Pepsin, 64, 65
 administration of, 259, 262
 determination of, 83
 by gelatin test, 50, 84
 by Jacoby-Solms method, 83
 by Mett test, 84
 by ricin test, 83
 in gastric disorders, 674
 hydrochloric acid and, 258, 259
 precautions in administering, 259
 unit, 84
 Pepsinogen and pepsin, examination for, 83
 relation of HCl to, 54, 83
 Peptic digestion, end products of, 54
 ulcer of esophagus, 360
 Peptids, 54
 Peptone preparations, 188
 food value of, 189
 Peptones, test for, 85
 Perforating gastric ulcer, 488, 509
 Perigastritis, 524
 causes of, 524
 diagnosis of, 525
 roentgenography in, 525
 forms of, 525
 hour-glass stomach in, 524
 prophylaxis of, 526
 symptoms of, 525
 treatment of, 526
 Perineal lacerations, nervous dyspepsia and, 419
 Perionitis alveolaris dentalis, 331
 treatment of, 332
 Perisigmoiditis, acute, 785, 789
 appendicitis and, 789
 chronic, 790
 diverticulitis and, 785, 789
 etiology of, 785
 symptoms of, 788
 treatment of, 790
 ulcerative, 789
 Peristalsis, effect of opium on, 274
 of intestine, 62. *See also* Intestinal peristalsis.
 of stomach, 51, 140
 in duodenal ulcer, 709
 Peristaltic nodes, 63, 561, 696
 restlessness, 782
 rush, 63
 stimulants, 172
 unrest of stomach, 392
 treatment of, 393
 waves, double, 139
 Peristaltic in stonic constipation, 667
 Peritoneal inflation in roentgenography, 150
 Peritonitis, circumscribed, 767
 Perityphilitis, 767
 Permeability of pylorus, 91
 Pernicious anemia, duodenal contents in, 110
 hookworm disease and, 811
 Pernicious anemia, mouth affections in, *III*
 Peroxids in intestinal diseases, 230
 Pessaries, hemorrhoidal, 825, 826
 Petrissage, 210, 213
 Petrolatum, liquid, in constipation, 582
 664
 in gastroenteroptosis, 582
 in rectal carcinoma, 834
 Petroleum jelly in intestinal stasis, 694
 Pharyngitis, acute, 346
 chronic, 346
 Pharynx, affections of, 346
 Phenacetin, occasional oral effects of, 301
 Phenol in gastric diseases, 272
 treatment of hemorrhoids, 823
 Phenolphthalein as a laxative, 286
 ring test for occult blood, 124
 test for total acidity of stomach contents, 79
 Phlegmonous esophagitis, 356
 gastritis, 457
 processes of tongue, 324
 Phlegmons of buccal fundus, 330
 Phosphorus stomatitis, 301
 Phrenoptosis, 560
 Physiology of digestion, 49
 Physostigmin as an evacuant, 283
 Piles, 821. *See* Hemorrhoids.
 Pilocarpin in gastric diseases, 271
 in sympathicotonia, 392
 vs. atropin, 271
 vs. epinephrin, 390
 Pineapple juice as a proteolytic, 263
 Pinworms, 803
 appendicitis and, 767
 Pituitary extract in flatulence, 703
 in hemorrhage, 517
 in ileus, 754
 in intestinal paresis, 284, 703
 in meteorism, 703
 Plaques of leukoplakia, appearance of, 319
 removal of, 320
 of lingua geographica, 323
 opales, 310
 syphilitic, in mouth, 310
 Plasmon, 192
 Pneumatosis (drum-belly), 403
 treatment of, 403
 Poikilocytosis in hookworm disease, 810
 Poisoning, alkali, fatality of, 357
 lavage in, 197
 lead, stomatitis from, 301
 mercurial, oral effect of, 299
 prophylaxis of, 300
 treatment of, 300
 phosphorus, stomatitis and abscess from, 301
 sulphuric acid, fatality of, 357
 Polychromatophus in hookworm disease, 810
 Polyneuritis, rice diet and, 157
 Polyp, mucous, 353

- Polypi, rectal, 835
 Polyposis recti, 836
 Polysaccharids, 184
 Postoperative obstructions, roentgenography in, 149
 Potassium chlorate in mouth diseases, 297
 Potato poultices, 250
 remnants in stool, 121
 Potatoes as a food for gastric patients, 111
 in intestinal diseases, 182
 Pot-belly in hookworm disease, 811
 Poultices, linseed, 250
 potato, 250
 Powder blowers, intra-intestinal, 238
 Pregnancy as a cause of gastroenteroptosis, 557
 as a remedy for gastroenteroptosis, 581
 gingivitis in, 303
 Priessnitz bandage, 250
 Proctidentia recti, 845
 Proctitis, 841
 chronic, 842
 diagnosis of, 841
 etiology of, 841
 hot water in, 842
 symptoms of, 841
 treatment of, 842
 surgical, 843
 symptomatic, 842
 Proctoclysis, 239
 apparatus, 240, 245
 continuous, Young's apparatus for, 242
 Elbrecht's heating apparatus for, 240, 242, 243
 use of thermos flask in, 242
 Proctoscope, Kelly's, 230
 Proctospasm, etiology of, 848
 symptoms of, 848
 treatment of, 849
 dilatation, 849
 galvanization, 849
 refrigeration, 849
 Prolapse of rectum, 845
 Propeptone, test for, 85
 Prosecretin, 57
 Proteal, 550
 Protectives in intestinal diseases, 279
 Protein, absorption of, 60
 examination of feces for, 117
 limitation of, in chronic pancreatitis, 627
 in food cures, 571
 milk-salt-cocca, 193
 preparations, animal, 187
 from eggs, 192
 from milk, 191
 vegetable, 191
 soluble, in stool, 121
 Proteolysis, HCl and, 259
 pepsin and, 259
 Proteolytic action of pineapple juice, 211
 Protogen, 192
 Protozoa in stomach contents, 92
 Pruritus ani, 851
 anal dilatation in, 855
 plugs in, 856
 bacterial vaccines in, 856
 Ball's operation in, 857
 calomel for itching of, 853
 diet in, 853
 dry, 852
 etiology of, 851, 852
 hot applications in, 854
 local and general conditions associated with, 852
 moist, 852
 ointments for, 853, 854, 855
 pathogenesis of, 851
 quinin and urea hydrochlorid in, 855
 Roentgen ray in, 856
 streptococci in, 852, 856
 thread worm and, 803
 tincture benzoin in, 853
 treatment of, 853
 ulceration in, 851
 of jaundice, 609
 Psychic initiation of gastric secretion, 52, 67
 Psychogenic nervous diarrhea, 680
 Psychotherapeutics in aerophagy, 402
 in akoria, 416
 in cardiospasm, 396
 in rumination, 406
 Ptosis and appendicitis, 768
 Ptyalin, action of, 50
 Ptyalism, 330
 Pumpernickel, 165, 184
 Pupillary symptom of pancreatic insufficiency, 626
 Purgative drugs, 284
 effect of mineral waters, 254, 255
 Purgatives, 282
 indications for, 282
 the mildest, 284
 Pus in intestinal contents, 122
 in stomach contents, 66
 Putrefaction of feces a peristaltic stimulant, 172
 Pyloric closure, cicatricial, lavage in, 198
 dilator, Einhorn's, 400, 485
 function, vertebral pressure and, 211, 405
 insufficiency, demonstration of, 96
 treatment of, 407
 obstruction, roentgenography in, 142
 patency, blue bead to demonstrate, 91
 duodenal bucket to demonstrate, 91
 stenosis, 96
 congenital, 510

- Retarded development in hookworm disease, 811
 Retention of gastric contents, massage in, 208
 Retropharyngeal abscess, 348
 Rhizotomy in treatment of gastralgia, 412
 Rib, fluctuating, 63
 Rice as an article of diet, 166
 diet and polyneuritis, 157, 167
 injured by "polishing," 166
 vitamin in, 157
 Roberts' oil enema, 225
 rectal dilator, 839
 Roborat, 191
 Roentgen fluoroscopy, manipulation during, 135
 in sigmoidal loops, 148
 Roentgen-ray examination, 135
 of esophagus, 136
 of intestine, 143
 of stomach, 138
 location of swallowed teeth, 377
 in pruritus ani, 856
 treatment of carcinoma, 550
 Roentgenography in appendicitis, 145, 147
 in atonic constipation, 661
 barium in, 145
 or bismuth in, 135
 bismuth in, 137, 138, 139
 in callous gastric ulcer, 141
 in cardiospasm, 137, 396
 in cecal volvulus, 147
 in cholecystitis, 150
 in colonic carcinoma, 145
 dilatation, 148
 stasis, 147
 in duodenal ulcer, 143
 in enlargement of liver, 149
 in enteroliths, 743
 in esophageal dilatation, 137
 spasm, 137
 in examination of appendix, 147
 of esophagus, 136
 of colon, 144
 of intestine, 143
 of stomach, 138
 in gall-bladder disease, 149
 in gastroenteroptosis, 564
 in ileal stasis, 144
 in intestinal carcinoma, 764
 distention and obstruction, 149
 spasm, 669
 stenosis, 148, 756, 757
 tumors by outlining spleen, 150
 in pancreatic carcinoma, 149
 cysts, 149
 in perigastritis, 525
 peritoneal inflation in connection with, 150
 in pyloric obstruction, 142
 in rectal constipation, 146
 Roentgenography in spastic constipation, 669
 showing acute splenic flexure, 146
 appendix, 145
 atony of stomach, 140
 carcinoma of esophagus, 138
 of stomach, 142
 cardiac stricture, 137
 cardiospasm, 137, 396
 cecum mobile, 146
 coloptosis, 146
 deformity of duodenal cap, 143
 deviation of colon, 146
 of sigmoid, 148
 dilatation of stomach, 140
 diverticulum of esophagus, 138
 form of colon, 144, 145
 of rectum, 146
 of stomach, 138, 139
 gallstones, 150
 gastric ulcer, 141
 gastropnoia, 139
 Hirschsprung's disease, 148
 hour-glass stomach, 142
 incompetency of ileocecal valve, 144, 148
 intestinal stenosis, 148
 motility of stomach, 51, 140
 position of colon, 145
 pyloric obstruction, 142
 salivary calculi, 328
 sigmoidal adhesions, 148
 diverticulitis, 148
 spastic constipation, 146
 tonicity of colon, 146
 tonus of stomach, 140
 in volvulus, 147
 Rosenberg's powder blower, 238
 rectal dilator, 840
 irrigation apparatus, 234
 Rosenheim's esophageal syringe, 354
 tube for stomach douche, 205
 Rose's adhesive plaster belt, 579
 Round-worm, 800
 chenopodium in treatment of, 802
 diagnosis of, 800
 intestinal perforation by, 801
 santonin in treatment of, 802
 symptoms of, 800
 treatment of, 801
 Rovsing's sign of chronic appendicitis, 772
 Rubecola, faucial and palatal effects of, 302
 Rub-off, the, 247
 Rumination, 105
 psychic treatment of, 406
 Russell's emulsion, 194

- Sahli's desmoid test for gastric secretion, 90
 glutoid capsule test of pancreatic function, 129
- Salicylates in gastric diseases, 272
- Salicylic acid in intestinal diseases, 280
 milk, 176
- Saline solutions, absorption of, 61
- Saliva, two-fold action of, 49
- Salivary calculi, 328
 digestion, 49
 ducts, diseases of, 327
 glands, actinomycosis of, 329
 diseases of, 328
 in general affections, 329
 enlargement of, 330
 syphilis of, 329
- Salivation from bromids, 301
 from iodids, 301
 from mercury, 299
 from paralysis of facial nerve, 320
 from scorbutus, 302
 total arrest of, 321
- Salomon's test for gastric carcinoma, 86
- Salpingitis and nervous dyspepsia, 419
- Salvarsan. *See* Arsphenamine.
- Salvatose, 188
- Sanatogen compared with milk, 192
- Santonin, effects of, 802
- Sarcinae in stomach contents, 92
- Sarcoma of alveolar processes, 346
 and carcinoma of stomach, differential diagnosis of, 554
 of esophagus, 364
 of intestine, 766
 of liver, 601
 of maxillae, 346
 of palate, 341
 of rectum, 834
 of stomach, diagnosis of, 554
 etiology of, 553
 pathology of, 553
 symptoms of, 553
 treatment of, 554
 of tongue, 341
- Scarlet fever, diseases of salivary gland and, 329
 the tongue in, 301
 red in gastric ulcer, 505
- Schmidt's and Strasburger's contribution to fecal analysis, 112, 172
 diet, 112
 incubator test for fermentation and putrefaction of feces, 116
 sublimate test for fecal pigment, 116
 test diet before fecal examination, 112
 for nuclei in feces, 126
- Schreiber's esophageal dilator, 370
- Scleroderma in mouth, 318
- Scleroma, 315
 treatment of, 315
- Scolecitis, 767
- Scolecoiditis, 767
- Scorbutic stomatitis, 302
- Scotch douche, 251
- Scurvy from vitamin deficiency, 158
- Sea baths, 256
- Sea-water in cholera morbus, 642
 dispensaries, 642
 in infantile gastroenteritis, 643
 in membranous enteritis, 657
 in nervous dyspepsia, 423
 technic of injection of, 424
- Seat worm, 803
- Secretin, 55, 57, 258, 259
- Secretion. *See* Gastric secretion and Pancreatic secretion.
- Secretory disorders of mouth, 321
 neuroses, 430, 440
- Sedatives, gastric, 268
 intestinal, 274
- Segmenting movements of intestine, 62
- Senator's esophageal dilator, 370
- Sennatin in atonic constipation, 667
- Sensory neuroses, 410
- "Sentinel pile" of anal fissure, 859
- Sepsis, oral, 290
- Serologic reactions in gastric carcinoma, 543
- Serum treatment of febrilis icterus, 588
 of trichinosis, 820
- Truncatek's, 532
- Sevetol, 194
- Sexual excesses as a cause of nervous dyspepsia, 419
- Shad-belly, 811
- Sialadenitis, 328
- Sialodochitis, 327
- Sialoliths, 327
- Sigmoid flexure, deviations of, shown by
 Roentgen ray, 148
 positional changes of, in gastroenteroptosis, 560
 redundant, 560
- Sigmoidal diverticula, 787, 788
 diverticulitis, 786, 789
 etiology and pathology of, 786, 788
 roentgenography in, 790
 symptoms of, 789
 inflammation, 785. *See* Sigmoiditis.
 origin of intestinal affections, 785
- Sigmoiditis, chronic, 644. *See* Intestinal catarrh, chronic.
 ulcerative, 730
- Sigmoidoscope, pneumatic (Strauss), 237
- Silver nitrate in gastric diseases, 267
 poisoning, oral effect of, 301
- Singultus gastricus, 408
 treatment of, 408
- Sinusoidal current in digestive disorders, 219
- Sippy's esophageal dilator, 369, 370
 treatment of gastric ulcer, 449
- Skin diseases in esophagus, 357
 in mouth, 316

- Sleep after eating, 171
 Sodium cacodylate in nervous dyspepsia, 425
 chlorid waters, 253
 Sodium-salvarsan, 534
 Somatine, 188
 Somatose, 187
 from milk, 192
 Sounds, diverticular, 376
 esophageal, 365, 367
 (noises), deglutition, 350
 Soups in putrefactive intestinal diseases, 178
 Spasms from vagotonia, 389
 Spastic constipation, 668. *See* Constipation.
 Spices in the diet, 167
 Splanchnoptosis, 557
 Spleen outlined by Roentgen ray, 150
 Splenoptosis, 569
 Sprue, 308
 Starch agar tubes, 102
 conversion, stages of, 50
 in the stool, 121
 Starck's diverticular sound, 376
 Steapsin, 58
 in feces, demonstration of, 125
 Grutzner-Gamgee test for, 126
 von Oefele test for, 126
 Steatorrhea, 127
 Stenoses, intestinal, roentgenographic appearance of, 148
 pyloric, 96. *See* Pyloric stenosis.
 test-diet stools in, 134
 Stercoral ulcers, 736
 Stockton's combined stomach tube and electrode, 218
 Stomach, absorptive power of, 56, 89
 atony of, 95. *See also* Atony of stomach, and Motor insufficiency.
 shown by roentgenography, 140
 bucket, Einhorn's, 70
 carbohydrate digestion in, 85
 carcinoma of, 537. *See* Carcinoma of stomach.
 cells and their function, 54
 condition of, in constipation, 185
 contents in achylia gastrica, 95
 acidity of. *See* Acidity of stomach contents.
 aspiration method of obtaining, 68
 in atony, 95
 bile in, 66
 blood in, 66, 86
 Weber's guaiac test for, 86
 Boas-Oppler bacillus in, 92
 Chase's stomach tube for obtaining, 206
 crystals in, 94
 determination of gastric juice in, 73
 Stomach contents, determination of
 reaction of, 75
 in diarrhea, 676
 in erosions of stomach, 97
 examination of, 66
 chemical, 74
 for enzymes, 83
 indirect, 88
 macroscopic, 68
 microscopic, 92
 filtration of, reason for, 78
 food remnants in, 96
 fractional analysis of, 78
 in gastric atony, 95
 carcinoma, 97
 dilatation, 95
 neuroses, 94
 ulcer, 97
 in gastritis, 95
 HCl free and combined in, 75-81
 in hyperacidity, 94
 in hypersecretion, 94
 indirect methods of analyzing, 88
 inspection of, 72
 lactic acid in, 82
 methods for obtaining, 68
 aspiration, 68
 expression, 68
 regurgitation, 71
 with stomach bucket, 72
 in motor insufficiency, 92, 95
 mould fungi in, 92
 mucous membrane in, 94
 normal acidity of, 78
 protozoa in, 92
 in pyloric insufficiency, 96
 stenosis, 96
 phenolphthalein test for total acidity of, 79
 quantitative analysis of, 76
 fractional, 78
 normal solutions for, 77
 phenolphthalein test in, 79
 Töpfer's method, 80
 quantity of, 73
 reaction of, 75
 retention of, 208
 sarcinae in, 92
 tests for HCl in, 75, 76
 for lactic acid in, 82, 83
 cowhorn, 389
 cysts of, 555
 digestive function of, 52
 Günzburg's test of, 89
 Sabli's test of, 90
 dilatation of, 95. *See* Dilatation of stomach.
 douche, 205
 Einhorn's, 206
 Rosenheim's, 205

- Stomach douche, Turck's, 206**
 electrization of, 215
 electrode, 216, 217
 and tube, Stockton's, 218
 Boas', 216
 Einhorn's, 217
 Lockwood's modification
 of, 217
 Wegele's, 217
 erosions of, 97, 521. *See* Erosions
 of stomach.
 fibroma of, 555
 fibromyoma of, 555
 form of, 139
 hemorrhage from, 511. *See* Hem-
 orrhage.
 hour-glass, 142, 524
 pseudo, 139
 hunger contractions of, 64
 hyperesthesia of, 413
 hypermotility of, 392
 lavage, 197
 lipoma of, 555
 massage, 208
 motor function of, 90. *See* Motor
 function of stomach.
 mould fungi in, 92
 movements of, 50
 shown by roentgenography,
 51, 140
 nerve supply of, 52
 neuralgia of, 410
 neuroses of, 387
 normal outline of, 138
 peristalsis of, 51
 peristaltic unrest of, 392
 waves, double, in, 139
 Roentgen-ray examination of, 138
 sarcoma in, 92
 sarcoma of, 553. *See* Sarcoma of
 stomach.
 syphilis of, 533
 tetany of, 485
 tonus of, 140
 shown by roentgenography,
 51, 140
 tube, 69, 70, 199, 200, 203, 204
 for aspiration, Chase's, 207
 and bulb, Aaron's, 69, 70
 and electrode, Stockton's, 218
 and funnel, 200
 Friedlieb's, 203
 introduction of, 202
 perforated (Rosenheim's), 205
 Rehfuss', 78
 Strauss', 204
 Turck's, 206
 use of, by patients, 202
 tuberculosis of, 536
 tumors of, 537, 555
 ulcer of, 488. *See* Ulcer, gastric.
 water-trap, 139, 558
 See also Gastric.
Stomach-ache, 414
Stomachic, condurango as a, 267
Stomachic, HCl as a, 260
 orexin as a, 267
Stomachics, action of, 266
Stomatitis, aphthous, 307
 treatment of, 308
 erysipelatos, 307
 treatment of, 307
 from bismuth, 300
 from gout, 303
 from influenza, 302
 from lead, 301
 from mercury, 299
 from phosphorus, 301
 from scorbutus, 302
 from variola, 302
 gangrenous, 305
 treatment of, 306
 hair-tongue and, 323
 in infantile scurvy, 303
 of lingua geographica, 323
 noma, 306
 treatment of, 307
 simple or catarrhal, 305
 treatment of, 305
 sprue, 308
 thrush, 309
 treatment of, 309
 ulcerosa, 306
Stools, pathologic, in diagnosis, 119.
 See also Feces, and Test-diet stool
 findings.
Strasburger's fermentation tubes,
 117
 method of separating bacteria from
 feces, 118
Strauch and the nuclei test of Adolf
 Schmidt, 126
Strauss' funnel for lactic acid test, 82
 sigmoidoscope, 237
 suction tube, 204
 test for lactic acid, 83
Streptococci, transmutation of, 291
Streptococcus-pneumococcus group of
 organisms, 293
 viridans, 292
Stricture of duodenum, 755
 symptoms of, 755
 of esophagus, 361, 383
 dilatation of, 364
 of ileum, 758
 of intestine, 755
 diet in, 759
 infrapapillary, 755
 lavage in, 759
 liquid petrolatum in, 760
 purgatives in, 760
 suprapapillary, 755
 treatment of, 758
 of jejunum, 756
 of large intestine, 757
 consequences of, 758
 etiology of, 758
 roentgenography in diag-
 nosis of, 757
 symptoms of, 757

- Stricture of rectum, 837. *See* Rectal stricture.
 of small intestine, 755
 roentgenography in diagnosis of, 756
- String test in cardiospasm, 396
 in diagnosis of gastric ulcer, 493
 in pylorospasm, 399
- Strongyloides stercoralis, 816
- Strongylus duodenalis, 806
- Struma at base of tongue, 339
- Strychnin as an aid to intestinal peristalsis, 284
 in diarrhea, 407
 in gastric diseases, 266
 in gastrogenic diarrhea, 677
 in pyloric insufficiency, 266, 407
- Subacidity, silver nitrate in, 268
- test-diet stool findings in, 131, 673
- Subcutaneous nutrition in motor insufficiency, 482
- Sublimate test for fecal pigment, 116
- Suction tube, 202
 with double bulb, 204
- Sugar in constipation, 185
 in diet of gastric patients, 167
 effect of, on gastric secretion, 167
- Sugars as aperients, 284
- Sulphur as a purgative, 285
- Sulphuric acid poisoning, fatality of, 357
- Suppuration shown by fecal examination, 122
- Surgery of stomach or intestine, lavage preceding, 197
- Surgical bladder from trematodes, 818
 kidney from trematodes, 818
- Swallowing sounds, 350
- Swedish manipulation in chronic constipation, 228
- Sympathetic nerve plexuses accessible by massage, 214
- Sympathicotonia, 388
 signs of, 391
- Syphilis and arteriosclerosis, 528
 of esophagus, 359
 of liver, 399
 of mouth, 309
 primary, 309
 treatment of, 310
 secondary, 310
 treatment of, 311
 tertiary, 311
 treatment of, 312
- of salivary glands, 329
- of stomach, 533
 diagnosis of, 533
 treatment of, 534
 general, 535
 specific, 534
- Syphilitic plaques in mouth, 310
 ulcers of intestine, 741
 of rectum, 843
- Syringe, esophageal, 354
- T**
- TABES, pyorrhea in, 333
- Tænia saginata, 794, 795
 solium, 794, 796
- Tampons, rectal, in chronic constipation, 230
- Tannalbin, 277
- Tannic acid in intestinal diseases, 277
- Tannigen, 277
- Tannocol, 277
- Tannoform, 277
- Tannyl, 277
- Tapeworms, 794
 aspidium (male fern) in treatment of, 797
 bothriocephalus latus, 794, 797
 castor oil in treatment of, 799
 diagnosis of, 796
 hemonolepis nana, 794, 798
 mode of infection with, 795
 pelletierin in treatment of, 799
 pumpkin seed in treatment of, 799
 symptoms of, 795
 tænia saginata, 794, 795
 solium, 794, 796
 treatment of, 797
- Tapotement, 209, 213
- Tea, effect of, on digestion, 169
- Teeth, carious, and phlegmon, 330
 details in care of, 294
 swallowed, located by Roentgen ray, 377
- Telangiectasia of mouth, 336
- Temperature of food in constipation, 185
- Terrell's treatment of internal hemorrhoids, 829
- Test breakfast, Boas', 67
 Ewald-Boas', 67
 diet and its administration, 67, 112
 to determine intestinal function, 112
 stool, examination of, 114
 chemical, 116
 macroscopic, 116
 microscopic, 115
 findings in achylia gastrica and subacidity, 131
 in atonic constipation, 133
 in chronic constipation, 661
 in duodenal ulcer, 133
 in dysentery, 133
 in enteritis membranacea, 133
 in gastric carcinoma, 131
 ulcer, 131
 in hyperacidity, 131
 in intestinal carcinoma, 134

- Test-diet stool findings in intestinal catarrh, chronic, 131, 132
 fermentative dyspepsia, 134, 678
 tuberculosis, 133
 in nervous diarrhea, 134, 681
 in spastic constipation, 134, 670
 in stenoses, 134
 in subacidity, 131, 674
 dinner, Riegel's, 68
 meal, Einhorn's, before examining duodenal contents, 101
 Leube's, before examining duodenal contents, 90
 normal, 118
 Schmidt's, before examining feces, 112
 in treatment, 179
 Tests for blood in feces, 123
 in gastric contents, 66, 86
 for carcinoma of stomach, 86-88
 for diastase, 127
 for dissolved protein in feces, 117
 for enzymes, 83, 102
 for fat in feces, 127
 for fecal pigment, 116
 for fermentation and putrefaction of feces, 116
 for gastric absorption, 89
 acidity, 78-81, 88-90
 function, 89, 90
 motility, 90, 91, 129
 secretion, 90
 for hepatic insufficiency, 592-593
 for hydrochloric acid, 75-81, 88-90
 for intestinal motility, 128, 129
 for lactic acid, 82, 83
 for lipase, 592
 for nuclei in feces, 128
 for pancreatic function, 126, 129, 624-629
 for pepsin and HCl, 84
 for peptone, 85
 for propeptone, 85
 for steapsin, 125
 string, 396, 399, 493, 710
 for trypsin, 125, 126
 for urobilin, 101
 for vagotonia and sympathicotonia, 591
 Tetany, gastric, 485
 lavage in, 196
 Thermos proctodysis apparatus, 242
 Thiouanin in pyloric stenosis, 484
 Thread test for gastric acidity, 86
 worm, 803
 anthelmintics in, 803
 calcification of, 803
 diagnosis of, 804
 Thread worm, diet in, 805
 disinfection during treatment of, 805
 enenata in, 805, 806
 life history of, 803
 symptoms of, 803
 treatment of, 805
 Thrombotic ulcers of intestine, 741
 Thrush, 309
 of esophagus, 361, 378
 treatment of, 309
 Thyroid extract in arteriosclerosis, 532
 Tobacco in gastric disease, 169
 Tongue, abscess of, 324
 affections, 322
 angioma of, 336
 carcinoma of, 343
 coating of, 322
 cysts, 338, 339
 endothelioma of, 340
 enlargement of (macroglossia), 326
 fibroma of, 335
 furrowed, 326
 geographic (lingua geographica), 323
 hair (lingua nigra), 323
 inflammation of (glossitis), 324, 325
 lipoma of, 335
 lymphangioma of, 337
 lymphoma of, 327
 malformation of, 322
 neuralgia of, 321
 pain (glossodynia), 321
 papilloma of, 340
 phlegmon of (acute diffused glossitis), 324
 sarcoma of, 341
 in scarlet fever, 301
 struma of, 339
 tuberculosis of, 313
 ulcers of, 305, 325
 Tonsil, hypertrophy of, 326, 347
 lingual, hyperkeratosis of, 327
 Tonsillitis as a cause of other infections, 292
 chronic, 347
 follicular, 347
 lingual, 326
 parenchymatous, 347
 suppurative, 347
 Tonus of digestive tract, fundamental or extrinsic, 64
 of stomach shown by Roentgen ray, 140
 Tooth pastes and powders recommended, 294
 Töpfer's method of quantitative analysis in determining gastric acidity, 80
 Toruna intestinum nervosa, 782
 symptoms of, 782
 treatment of, 782
 Toxemia and arteriosclerosis, 529
 and oral sepsis, 290
 of ileus, 747
 intestinal, 603

- Toxic gastritis**, 455
 products of bacteria conditioned
 on culture medium, 684,
 685
 of intestinal putrefaction, 686
- Trematodes**, 816
 biliary calculi from, 818
 cirrhosis from, 818
 dysentery from, 818
 in portal vein, 817
 somatic habitat of, 817
 surgical bladder from, 818
 kidney from, 818
 treatment of, 818
- Trichina spiralis**, 818, 819
- Trichinae** in intestine, 818
 in muscle, 819
- Trichinosis**, diagnosis of, 820
 eosinophilia in, 819
 how to prevent, 160
 raw meat and, 160
 symptoms of, 819
 treatment of, 820
 with immune serum, 820
- Trichocephalus dispar**, 814
- Trophic disorders** of mouth, 321
- Tropon**, 188
- Trunccek's serum**, 532
- Truss**, Esmarch's rectal, 847
- Trypsin**, 57
 in feces, Gross' casein test for, 125
 Muller-Schlect test for, 125
 Schmidt's nuclei test for, 126
- Trypsinogen**, 58
- Tube**, colon, 220
 duodenal, Einhorn's, 98
 Gross', 100
 Kuhn's, 98
 Jutte's, 100
 Palefski's, 100
 gastroduodenal, Rehfuss', 78
 irrigating, Rosenberg's, 234
 Wolbarst's, 234
 Zweig's, 233
 Leiter's, 250
 rectal, 221
 stomach, Aaron's improved, 68-70
 Chase's, 207
 Friedlieb's, 203
 Rehfuss', 78
 Rosenheim's, 205
 Strauss', 204
 Turk's double flow, 206
 with electrode, Stockton's, 218
 with funnel connection, 200
- Tubercle bacilli** in feces, 122
 gastric juice and, 737
- Tubercular abscess** of pharynx, 348
 intestinal ulcers, 737
- Tuberculosis** of cecum, 740
 of esophagus, 359
 of intestine, 737
 test-diet stool findings in, 133
 of liver, 600
 of mouth, 312
- Tuberculosis** of stomach, 536
 of tongue, 313
- Tumors**, benign, of esophagus, 362
 of gall bladder, 614
 of intestine, 766
 of liver, 602
 of maxilla, 345
 of mouth, 335
 of stomach, 555
 of pancreas, 633
 malignant. *See* Carcinoma, Sarcoma, etc.
- Turck's double-flow stomach douche**, 206
- Tympanites**, 698
- Typhilitis stercoralis**, 736
- Typhoid carriers**, duodenal contents of, 110
 fever, danger of acidosis in diet of, 716
 diet, carbohydrates in, 716
 fat in, 716
 high caloric, 714
 need of sodium chlorid in, 716
 starch in, 716
 diseases of salivary glands and, 329
 duodenal contents in, 110
 Houghton's diet table for, 717
 palatal ulceration in, 302
 recrudescences as affected by diet, 714
 vegetable soup in, preparation of, 717
 ulcers, 713
- U**
- UFFELMAN'S test** for lactic acid, 82
- Ulcer**, alveolar, 321
 callous, roentgenologic diagnosis of, 141
 colitic, dry treatment of, 237
 duodenal, 705
 affecting form of stomach, 144
 age incidence of, 710
 appendicitis and, 708
 blood test in diagnosis of, 709
 cholelithiasis and, 708
 chronic cholecystitis and, 708
 constipation and, 708
 comfort posture of patient in, 709
 complications of, 710
 diagnosis of, 143, 707
 by posture, 709
 roentgenographic, 143, 710
 by silk string test, 710
 diet in, 711
 duodenal contents in, 110
 etiology of, 705
 fecal examination in, 133, 709
 gastric secretion in, 709

- Ulcer, duodenal, hemorrhage from, 707
 marasmus and, 710
 melena of infants and, 710
 pain of, 708
 perforated, 708
 perforating, an indication of, 144
 peristalsis in, 144
 polycythemia in, 710
 prognosis of, 711
 sex incidence of, 710
 symptoms of, 706
 test-diet stool findings in, 133
 treatment of, internal, 711
 surgical, 712
 experimental, 706
 gastric, 97, 488
 age incidence of, 489
 antilytic serum in, 506
 appetite in, 492
 bacterial vaccines in, 506
 bismuth in, 504
 subnitrate in, 266, 504
 callous, roentgenography of, 141
 complications of, 493
 diagnosis of, 493
 by string test, 493
 duodenal alimentation in, 500
 effect of healing of, 490
 etiology of, 489
 frequency of, 489
 from arteriosclerosis, 528
 hemorrhage from, 491
 incidence of vomiting in, 491
 incision in, 139
 localization of pain in, 491
 mud-baths in, 257
 olive oil in, 506
 pathology of, 488
 perforating, 488
 diagnosis of, 492
 roentgenography of, 181
 perforation from, 492, 509
 prognosis of, 494
 prophylaxis of, 495
 in pruritus ani, 851
 pyloric stenosis in, 509
 roentgenography of, 141
 scarlet red in, 505
 sequela of, 493
 sex predisposition to, 489
 silver nitrate in, 505
 stomach contents in, 97
 subphrenic abscess following perforation in, 509
 surgery in, 507
 symptoms of, 490
 test-diet stool findings in, 131
 treatment of, antigenic (by vaccines), 506
 Lenhart's, 498
 Leube-Ziemssen, 495
 medicinal, 503
 Sippy, 499
 Ulcer, gastric, treatment of, surgical, 507
 vomiting in, 491
 jejunal, 712
 palatal, 321
 peptic, 488
 perforating, demonstrated by Roentgen ray, 141
 round, 488
 of tongue, decubital, 325. *See also* Ulcers.
 Ulcerative colitis, 730
 enteritis, 730
 peri-sigmoiditis, 789
 sigmoiditis, 730
 Ulcerocarcinoma, 541
 Ulcers, dysenteric, 719. *See* Dysentery.
 of esophagus, 358
 intestinal, 705, 713, 719, 730, 737
 catarrhal and follicular, 730.
 See also Enteritis
 diet in, 172
 embolic, 741
 syphilitic, 741
 thrombotic, 741
 tubercular, 737. *See* Intestinal tuberculosis.
 of palate, 304
 of rectum, 843
 anodynes in, 844
 diagnosis of, 844
 gonorrheal, 843
 surgery of, 845
 syphilitic, 843
 treatment of, 844
 tubercular, 844
 stercoral or decubital, 736
 tibial, in hookworm disease, 811
 of tongue, 305
 typhoid, 713. *See* Typhoid fever.
 See also Ulcer.
 Ulcus rotundum duodeni, 705
 ventriculi, 488
 Umbilical dyspepsia, 428
 Uncinaria americana, 809
 duodenalis, 806
 Uncinariasis, 806. *See* Hookworm disease.
 Urethritis from trematodes, 818
 Urobilin and urobilinogen in duodenal contents, 101, 110
 Schlesinger's test for, 101
 Urobilinuria from disease of liver, 593
 Urticaria in mouth, 318
 Uvula, carcinoma of, 344
 Uzara, 276

VACCINE treatment of chronic intestinal catarrh, 649
 of enteritis membranacea, 656
 of gastric ulcer, 506
 of pruritus ani, 856
 of ulcerative colitis, 735

- Vaccine treatment of ulcerative enteritis,** 735
Vaccinia in mouth, 302
Vagotonia, 389
 and mucous colitis, 653
 and sympathicotonia, 388
 medical treatment of, 391, 656, 658
 signs of, 390, 391
Vagus and sympathetic, coördination of, 781
 gastric secretion and, 54
Varicella, oral localization of, 302
Variola, diseases of salivary glands and, 329
 in esophagus, 357
Varicellous stomatitis, 302
Vasomotor disorders of mouth, 321
Vegetable milk, 194
 protein preparations, 191
Vegetables in constipating diet of intestinal diseases, 179
 green, in diet of gastric patients, 167
Vegetative nervous system, 387, 781
 diagnosis of disturbances of, 390
 spastic constipation and, 668
Verneuil's dilatation treatment of hemorrhoids, 827
Vertebral massage in peristaltic unrest of stomach, 393
 pain in gastric ulcer, 491
 pressure and pyloric function, 211, 405
 symptoms of pancreatic disease, 827
Vibration following massage, 214
Vitamin, 157
 deficiency, effects of, 158
 foods rich in, 158
 growth-promoting, 159
 in milk, 157
 necessity of, 157
 in rice, 157
 susceptibility of, to heat and alkalis, 158
 three kinds of, 158
Volvulus of cecum diagnosed by Roentgen ray, 147
 hemorrhage from, 747
 massage in, 749
 origin of, 742
 of sigmoid, 748
Vomiting in gastric ulcer, 491
 hysterical, electricity in, 215
 idiopathic, 403
 of intestinal stricture, 755
 lavage in, 197, 198
 to prevent, 197
 nervous, 403
 treatment of, 404
 of peritonitis, lavage in, 198
 of pregnancy, blood transfusion in, 404
Vomiting of pregnancy, corpus luteum in, 405
 electricity in, 215
 epinephrin in, 404
 vertebral percussion in, 405
Von Oefele's test for steapsin in feces, 126
- W**
- WALES bougie,** 733
Warm entire pack, 248
Water in the diet, 168
 effect of, on gastric secretion, 168
 hot, before meals, 168
 mineral, in gastric diseases, 168
 retention of, in stomach, 168
 in therapeutics, 247
"Water way" in stomach, 52
Water-trap stomach, 139, 558
Waters, alkaline carbonated, 253
 bitter, 254
 chlorin, 252
 ferruginous, 254
 sodium chlorid, 253
Weber's guaiac test for blood in gastric contents, 86
Wegele's stomach electrode, 217
Weill's disease, 587
Wet rub, the, 247
Whey in the diet, 164
Whipworm, 814
 treatment of, 815
Whortleberry as an astringent article of diet, 177
Williams' hemorrhoidal electrode, 831, 832
Wohlgemuth's antidiabetic diet in pancreatic disease, 630
 test for diastase in feces, 127
Wolbarst's rectal irrigation tube, 235
Wolff-Junghans test for gastric carcinoma, 87
Worm carriers, 813
Worms, 794. *See* Tapeworm, Round worm, Thread worm, Hookworm, Whipworm, Anguillula, Trematodes, and Fricluna.
 appendicitis and, 767
 eosinophilia and, 800
 maggot, 804
 nervous dyspepsia and, 419
Wormseed, American, as a substitute for santalin, 802
- X**
- XEROPHTHALMIA from vitamin deficiency,** 158
Xerostomia, 321

Y

YAGITA'S method of diagnosing tape-
worm, 790

Yeast in chronic constipation, 606
as an intestinal disinfectant, 281

Yoghurt, 165
advantages of, 165
from tablets, 165
in gastric diseases, 165
in intestinal toxemia, 692
kefir, koumiss and sour milk,
comparison of, 165
life-prolonging effect of, 165

Yoghurt, nutritive value of, 164
Young's apparatus for continuous
proctoclysis, 242

Z

Zweig's ice-bag for painful hemor-
rhoids, 824

oil emulsator, 223
rectal electrode, 231
irrigating tube, 233

Zymogens, 53



LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

--	--	--

1000

L801 Aaron, C.D. 97067
All Diseases of the
1921 digestive organs.

[illegible]

